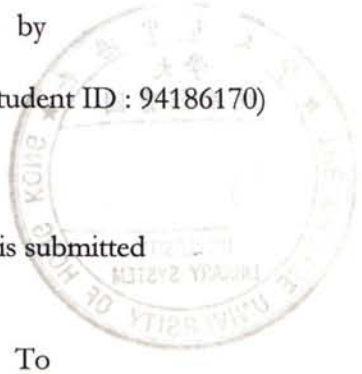
VARIATIONS OF THE COELIAC ARTERY AND HEPATIC ARTERY ORIGINS AND THEIR IMPORTANCE IN SELECTIVE INTERNAL RADIATION THERAPY

by

Ho Wai-chun (Student ID : 94186170)



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This thesis is dedicated to my daughter and my husband,
Charlotte and Anthony

腹腔血管和肝血管的種種差異
及其對
動脈插管式的選擇性內部放射線療法的影響

摘要

目的： 解剖學上的文獻經常提及到腹腔血管有很多差異；對于未能用手術切除的肝細胞癌(HCC)，動脈插管式的選擇性內部放射線療法是一種有效的療法。成功的超選擇性血管導引是這種療法的重要原素,而腹腔血管的種種差異直接影響這種療法的步驟。本研究旨在探討各種腹腔血管差異的頻率及兩性分別；血管差異對動脈插管式的選擇性內部放射線療法的影響及血管差異會否導致肝細胞癌亦包括在本研究內。 材料及方法： 本研究收集了 276 個個案，其中有 166 個 HCC 病例，其餘 110 個則爲非 HCC 病例。所有血管造影術均由同一位放射科醫生執行，採用常規 Seldinger 法及導引過程在 X 光儀器監察下進行。在Philips V3000的檢視台分析所有DSA造影圖像，紀錄所有腹腔血管及其分支的進口，再計算每一種進口的差異的頻率。研究的血管包括，腹腔血管、互通肝動脈、正肝動脈、右肝動脈、中肝動脈、左肝動脈、胃＋二脂腸動脈、右胃動脈、左胃動脈、及脾動脈。 結果： 各種腹腔血管差異的頻率用圖表方式紀錄在案。每個動脈進口的最高發生率均與解剖學文獻上所提供的『經典』動脈分佈圖一致。發生率最高的計有：腹腔血管的進口是大動脈(98.6%)、互通肝動脈的進口是腹腔血管(97.8%)、正肝動脈的進口是互通肝動脈(80.4%)、右肝動脈的進口是正肝動脈(80.4%)、中肝動脈的進口是右肝動脈(44.6%)、左肝動脈的進口是正肝動脈(77.5%)、胃＋二脂腸動脈的進口是互通肝動脈(93.1%)、右胃動脈的進口是正肝動脈(42.0%)、左胃動脈的進口是腹腔血管(94.6%)及脾動脈的進口是腹腔血管(99.3%)。這研究計算出72%研究個案的動脈分佈是有最少一個差異，餘下 28%則沒有任何差異，而 55.4%例的肝動脈血供是與『經典』動脈分佈圖吻合。這研究顯示腹腔血管的差異是沒有性別的不同；另外本研究闡述及分析各種差異對動脈插管式的選擇性內部放射線療法的影響。 結論： 本研究肯定了各種腹腔血管的解剖上差異，最重要的是能因應肝動脈血供及腹腔血管的種種差異去設計選擇性內部放射線療法，腹腔血管的差異並不會防礙選擇性內部放射線療法的使用。初步推斷，歐洲裔比亞洲裔人種有更多的血管差異，而血管的差異並不是導致肝癌的其中原因。

The Chinese University of Hong Kong

Abstract

VARIATIONS OF THE COELIAC ARTERY AND HEPATIC ARTERY ORIGINS AND THEIR IMPORTANCE IN SELECTIVE INTERNAL RADIATION THERAPY

by Ho Wai-chun

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Anatomic variations of the coeliac axis and its branches are known to be present in human as stated in anatomy textbooks. Trans-catheter Selective Internal Radiation therapy is a treatment of choice for the management of inoperable hepatocellular carcinoma. The success of this treatment procedure relies primarily on the superselective catheterization of the hepatic artery (or arteries) for delivery of ^{90}Yttrium microspheres and this is influenced by the vascular variations present in different subjects if any. This study was conducted from the point of view of a therapeutic interventionalist (instead of an anatomist) to determine the frequency of variants of the coeliac axis and its branches and presence of gender difference in Hong Kong Chinese. The influence of anomalies upon ^{90}Yttrium Selective Internal Radiation therapy and the possibility that arterial variation may be a predisposing factor to hepatocellular carcinoma was also studied.

276 subjects were recruited with 166 subjects confirmed as having HCC and 110 subjects without HCC. All arteriograms were performed by the same radiologist using the Philips V3000 Digital Subtraction Angiographic (DSA) system. Seldinger technique was employed and catherization was monitored under

fluoroscopic control. Serial arteriograms with predefined filming sequences were obtained. All resultant arteriograms were viewed on the viewing console and the arterial patterns were analysed. Origins of the coeliac axis and its branches were recorded and the percentage frequency were later calculated. The vessels studied included coeliac axis, common hepatic artery, proper hepatic artery, right hepatic artery, middle hepatic artery, left hepatic artery, gastroduodenal artery, right gastric artery, left gastric artery and splenic artery.

Variations of the coeliac axis and its branches in Hong Kong Chinese were documentated and their corresponding percentages charted. Highest occurrence of individual vessel origin was in conformity with the classical pattern mentioned in anatomy textbook. Predominant vessel origins were coeliac axis from aorta (98.6%), common hepatic from coeliac axis (97.8%), proper hepatic from common hepatic artery (80.4%), right hepatic from proper hepatic artery (80.4%), middle hepatic from right hepatic artery (44.6%) or from left hepatic artery (32.6%), left hepatic from proper hepatic artery (77.5%), gastroduodenal from common hepatic artery (93.1%), right gastric from proper hepatic artery (42.0%), left gastric from coeliac axis (94.6%) and splenic from coeliac axis (99.3%). 72 % of subjects had at least one variant and 28% had none. 55.4% of subjects had the liver arterial supply presented as the classical pattern. No gender difference was noted in the arterial variations. The influence of the variations of the coeliac and its branches upon the technique of ^{90}Yttrium microspheres delivery is extensively discussed and illustrated.

In Hong Kong Chinese, coeliac axis and its branches did present a spectrum of variations which required conscientious modifications during Selective Internal Radiation therapy for hepatocellular carcinoma. This therapeutic procedure was feasible in every individual needing it despite the presence of arterial variation. Apparently, arterial variations were more extensive in Europeans than in Hong Kong Chinese. Gender did not predispose to particular arterial patterns and

presence of arterial variation was not a pre-disposing factor for hepatocellular carcinoma.

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GLOSSARY OF ABBREVIATIONS

^{131}I	^{131}Iodine
^{90}Y	^{90}Yittrium
^{90}Y-SIR	Intraarterial infusion of ^{90}Y microspheres for SIR therapy
^{99m}Tc	^{99m}Technetium
Bq	Becquerel
CT	Computed tomography
HAG	Hepatic arteriography
HCC	Hepatocellular carcinoma
JHC	Joint Hepatoma Clinic
KeV	Kilo-electron-volt
MBq	Mega-Becquerel
MeV	Mega-electron-volt
mCi	Milli-Curie
PWH	Prince of Wales Hospital
SIR	Selective Internal Radiation
Tc-MAA	^{99m}Technetium-Macroaggregated-Albumin Scan
T/N ratio	Tumour to non-tumour ratio
USG	Ultrasonogram

Vessels:

Ab	Absent
ACC fr GD	Accessory from gastroduodenal artery
ACC fr LG	Accessory from left gastric artery
ACC fr PH	Accessory from proper hepatic artery
ACC fr SMA	Accessory from superior mesenteric artery
Ao	Aorta
BIF of CH	Bifurcation of common hepatic artery
BIF of Coeliac	Bifurcation of coeliac axis
BIF of PH	Bifurcation of proper hepatic artery
CH	Common hepatic artery
Coeliac	Coeliac axis
GD	Gastroduodenal artery
LG	Left gastric artery
LH	Left hepatic artery
MH	Middle hepatic artery
PH	Proper hepatic artery
RG	Right gastric artery
RH	Right hepatic artery
RH & LH	Both right and left hepatic arteries
SMA	Superior mesenteric artery

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I would like to thank Professor Metreweli[1] for his thought promoting advice during the presentation sessions of this undertaking. In particular, special thanks to Dr. Michael Chan[2] who had performed all the angiography and had given conscientious guidance in the preparation of this thesis. To Dr. Stephen Ho[3] I am indebted for helping me understand the ^{90}Yttrium and its use in the Selective Internal Radiation therapy for hepatocellular carcinoma. Lastly, I must thank my husband, Anthony, for being the personal engineer for my personal computer system. Without him, this manuscript would not have been born.

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STATEMENT OF ORIGINALITY

This study was done in the Prince of Wales Hospital, from Janurary of 1995 to December of 1997.

All the data within this study were collected by the author herself. The data cannot be used for other academic/scientific paper without the author's consent.

Chapter 1

INTRODUCTION

Hepatocellular carcinoma presents an enormously difficult clinical problem. Patients with hepatoma represent a rather special physiologic situation - perhaps more special than any other adult malignancy. Hepatomas often arise in the background of chronic liver disease - such as post-necrotic cirrhosis or chronic hepatitis. Surgery is the preferred treatment when the tumour is confined to a resectable segment or lobe. Its prognosis is extremely poor with a median survival of less than three months (H.K. Cancer Registry, 1993). Most cases are considered inoperable at the time of diagnosis due to extreme tumour extension and accompanying advanced cirrhosis. Disease localised to one lobe of the liver without any major vascular involvement or extrahepatic disease is usually resectable provided the liver reserve is good. Portal vein or hepatic vein invasion precludes the patient from surgery. (Leung Wai-Tong, 1995) About 90% of patients are not suitable for resection at the time of presentation (Shiu *et al.*, 1990). At the Joint Hepatoma Clinic of the Prince of Wales Hospital of Hong Kong, locoregional treatment using intra-hepatic-arterial chemotherapy, chemoembolisation and selective internal radiation with Lipiodol-^{131}I or ^{90}Y microshperes is currently employed for the treatment of inoperable HCC (Leung *et al.*, 1994). At present, studies show that intraarterial ^{90}Y microspheres given through an angiographic catheter is a feasible treatment for non-resectable HCC (W.Y.Lau *et al.*, In press).

Percutaneous trans-catheter ^{90}Y-SIR treatment is a hybrid of the advancement in at least three areas, viz. angiographic technique, digital imaging technique and radiation oncology. The first documented arteriogram was performed in January of 1896. Through decades of improvement till 1953, Sven Ivar Seldinger of

Sweden introduced the Seldinger technique of inserting a catheter into a blood vessel through a skin puncture. This marked the birth of modern angiography and interventional radiology (W.C.G. Peh, 1996, p.90) . In the 1980s, digital vascular imaging came into routine clinical use and with digital subtraction, maximum information can be extracted with elimination of the uninformative bony structures (W.C.G. Peh, 1996, p.88). Furthermore, real time high resolution fluoroscopy is a prerequisite to every interventional procedure, especially vascular interventional procedures. Röntgen's discovery of x rays in 1895 marked a new era in the medical field. X rays were used not only in diagnosis, they had applications in therapy as well. Shortly after the discovery of x rays, the first cancer treatment using x rays was reported in January of 1896 (W.C.G. Peh, 1996, p.93). In the beginning of the twentieth century, radionuclides started to be used in cancer treatment. In the nineteen eighties, 90-Yittrium shed light to the treatment of the difficult tumour, hepatocellular carcinoma. In the early nineties, 90-Yittrium microspheres were infused under fluoroscopy into the hepatic artery through an arterial porta-cath which was implanted during laparotomy (W.Y.Lau *et al.*, 1994). In the mid nineties, trans-catheter treatment came to light because of technical advances in digital vascular imaging and vast improvement in catheter and guidewire technology. Also, the partition model has made Selective Internal Radiation therapy using 90-Yittrium microspheres safe and repeatable without laparotomy (S.Ho *et al.*, 1997).

The first step to successful trans-catheter Selective Internal Radiation treatment is one or more successful superselective catheterizations. Finding a good match between the selective catheter, the guidewire, and the vascular anatomy is the secret to successful superselective catheterization (Abrams, 1997, p.155). Thus, arterial supply to the liver and any variants of the coeliac axis and its branches dictate the catheter placement for infusion of the ^{90}Y microspheres and any necessary modifications during the 90-Yttrium Selective Internal Radiation treatment procedure.

Constituents of the coeliac axis and the upper segment of the superior mesenteric artery vary to such an extent that any classification of the axis is purely arbitrary and, accordingly, will differ with the viewpoint adopted by the author (Michels, 1955). The fact that arterial variations do exist was well documented. Haller, the great Swiss physiologist, emphasized 200 years ago the need for an atlas on the variational anatomy of the coeliac artery which supplies the abdominal organs. Thompson, of Canada (1933), expressed that if anatomic variations are worth knowing at all, they are worth knowing as accurately as possible. The most recent large scale study of the abdominal arterial variations was done by Michels in 1955. In the study, 200 cadavers were dissected and statistical analysis of the blood supply of the liver and gallbladder was done. In the 1966, Michels made another publication based on the previous 200 dissections on the description of ten basic types of hepatic blood supply, the sources of singly and dual cystic arteries and of aberrant left and right hepatics and twenty-six collateral pathways to the liver (Michels, 1966). The studies were based on dissections of cadavers and from the anatomist's or the surgeon's point of view.

In this study, the classification is from the point of view of a radiological interventionist who delivers the 90-Yittrium microspheres mixture to the hepatocellular carcinoma. The vessels studied are the coeliac axis, common hepatic artery, proper hepatic artery, right hepatic artery, middle hepatic artery, left hepatic artery, gastroduodenal artery, right gastric artery, left gastric artery, and the splenic artery. The origins of these vessels dictate the catheter tip placement for delivering the radioactive microspheres mixture to the tumour and at the same time preventing gastritis caused by passage of radioactive microspheres into the vessels supplying the stomach and duodenum.

In this thesis, after the introductory chapter, some basic principles are described in chapter two. These include anatomy and blood supply of the liver, normal and variant arterial anatomy of the coeliac axis, management of hepatocellular carcinoma and knowledge of the 90-Yttrium Selective Internal Radiation therapy for inoperable hepatocellular carcinoma. The classification method used in this study are defined in chapter three. The objectives of this study are then stated in chapter four, and the materials and methods are described in chapter five. In chapter six, the results are presented with the bar charts showing the frequency of the vessel origins, and arteriograms are illustrated. Chapter seven is on result analysis, and the influence of the arterial variants on the 90-Yttrium Selective Internal Radiation treatment procedure is discussed. A final conclusion is given in chapter eight.

Chapter 2

BASIC PRINCIPLES

2.1 The liver

2.1.1 A vital organ

The liver is the largest organ in the body, weighing 1.2-1.5 kilogram (Macleod J, 1978). It occupies most of the right upper quadrant and epigastrium of the abdominal cavity (figure 2.01).

Although the liver is considered to be a structural part of the digestive system, it performs many functions not directly concerned with digestion. It has phagocytic cells (Kupffer's cells) which engulf bacteria and other foreign particles in the blood. Kupffer's cells are also very efficient at removing immune complexes from the blood. Thus the liver is capable of preventing undesirable immunological reactions. It is the most important organ for maintenance of normal blood glucose concentration. The synthesis of many proteins with their export into the blood is a major function of the liver. The liver is also responsible for amino-acid metabolism prior to their interconversion and oxidation. Urea synthesis occurs solely in the liver. The liver is quantitatively the most important organ for drug metabolism and it metabolizes about 90 percent of all alcohol ingested. A major digestive function of the liver involves the production and secretion of bile.

The liver actually exerts a number of important influences on nutrition : its bile secretion is essential in the digestion of fat, it stores glycogen, it stores vitamin A, B_{12}, D, E, and K, and it is involved in the metabolism of proteins, fats, and carbohydrates.

Apart from the above mentioned, the liver is the main heat producing organ of the body due to its high metabolic rate. Other functions of the liver include blood cell formation (hematopoiesis), coagulation, and detoxification.

2.1.2 Segmental anatomy of the liver

The liver has a right lobe and a left lobe. The right lobe is larger, and it contains the quadrate and the caudate lobe(Macleod J, 1978). A line from a point to the left of the gallbladder fossa anteriorly and inferiorly to the vena cava dorsally divides the liver into anatomic right and left lobes. No visible anatomic landmark helps to distinguish right from left lobes. The umbilical fissure and the falciform ligament divide the left lobe into medial and lateral segments, The right lobe contains anterior and posterior segments, although there are no surface markings to delineate the right lobe segments (R. Scott Jones, 1990). (Figure 2.02, figure 2.03)

The segmental anatomy of the liver is based on the intrahepatic distribution of the portal trinity (portal vein, bile duct, hepatic artery) and its divisions. There are eight segments and segment I is the caudate lobe of the liver. The caudate lobe is an autonomous segment receiving branches of the portal trinity from both right and left sides (Bernard, Jamieson, Starzl, 1993). The other segments, segment II to segment VIII are shown schematically in figure 2.04.

Figure 2.01 The position of the liver in the abdominal cavity.
(From Foundations of Anatomy and Physiology by Ross & Wilson)

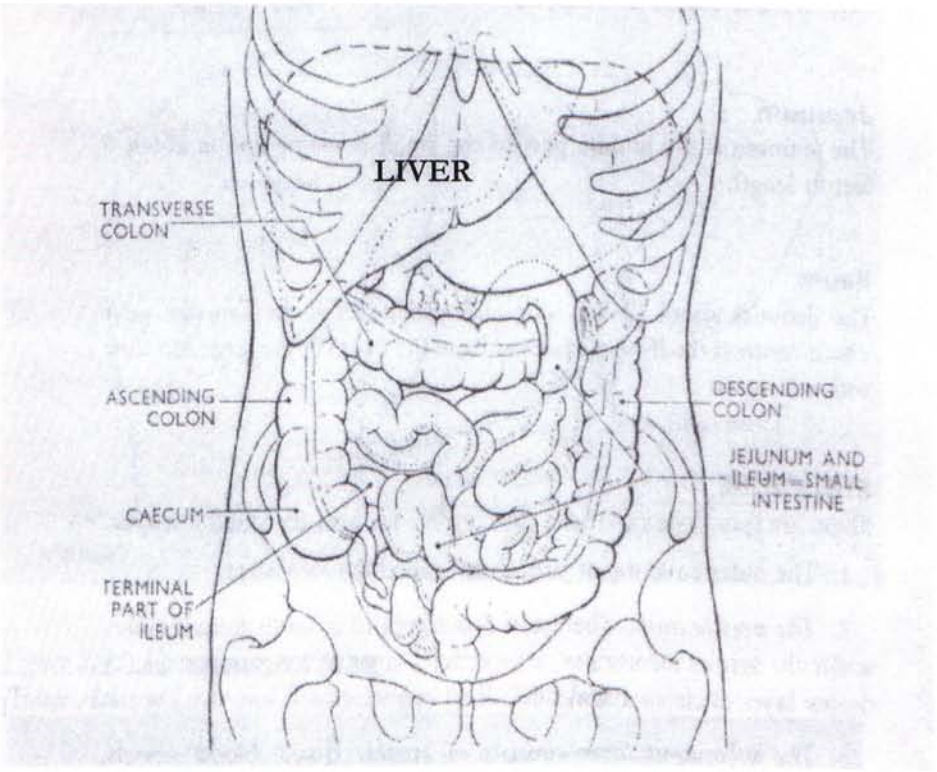


Figure 2.02 Ventral view of the liver
(From Atlas of Liver and Biliary Surgery by R. Scott Jones)

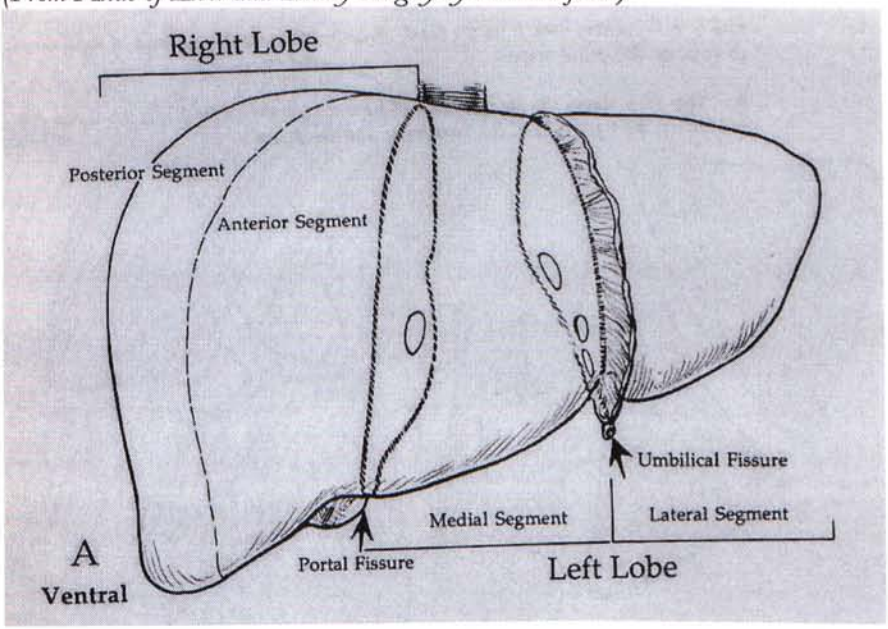


Figure 2.03 Dorsal view of the liver
(From Atlas of Liver and Biliary Surgery by R. Scott Jones)

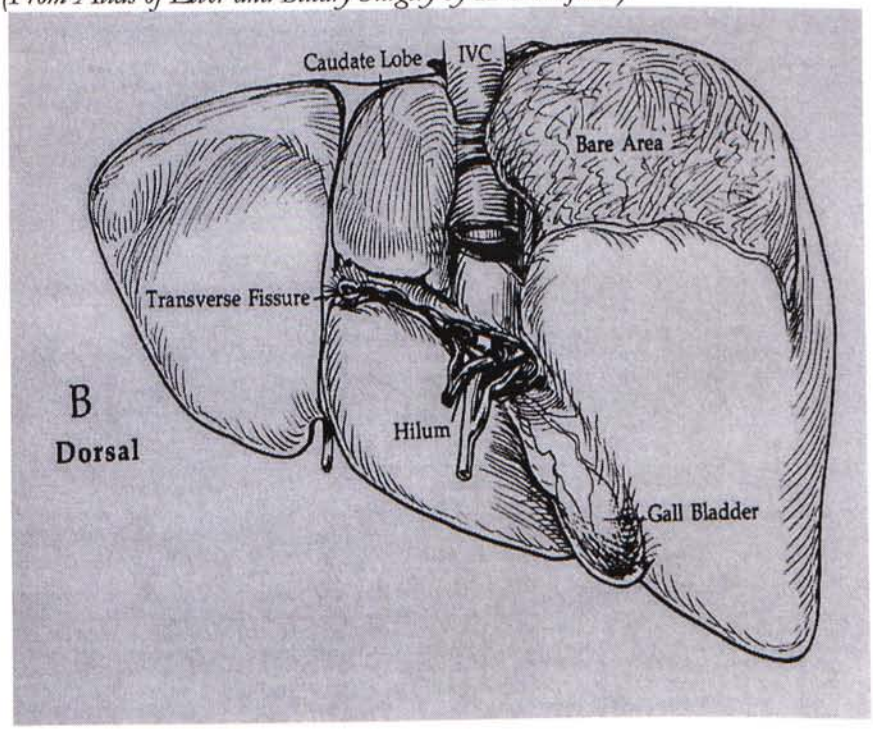
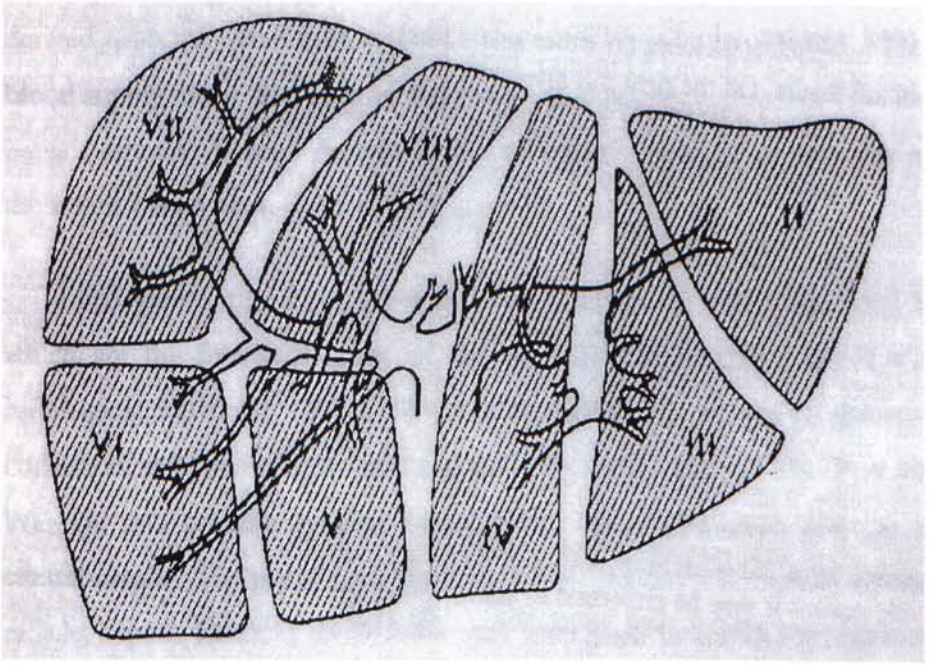


Figure 2.04 Segments of the liver.
(From Modern Operative Techniques in Liver Surgery by Launois, Jamieson & Starzl)



2.1.3 Vascular anatomy of the liver

Much of the importance of the liver is largely due to its dual blood supply derived from the gut as well as from the systemic circulation. About 75% of the blood supply to the liver is from the portal veins and the remaining 25% from the hepatic arteries. Venous drainage is via the hepatic veins which drain directly into the inferior vena cava. (Ross & Wilson, 1973)

Histologically, the liver parenchyma is made up of small polyhedral lobules which are the functional units of the liver. (See Appendix I for drawings of histological anatomy) Each lobule is approximately 1 mm in diameter and consists of columns of liver cells and sinusoids with a central vein. Pervading the liver are two separate systems of pipelines : the portal tracts and the hepatic central canals. The portal tract lies between the lobules and contain a portal vein radicle, hepatic arteriole, and bile duct. The lymphatic channels are also located in the portal tracts. The hepatic central canals contain radicles of the hepatic vein into which the sinusoids drain arterial and portal venous blood.

2.2 Blood supply of the liver

2.2.1 Arterial supply

The hepatic arterial supply has many variations. Details of the common variations are discussed in the next section. The classical presentation is shown in figure 2.05 with the common hepatic artery arising from the coeliac axis. The common hepatic artery, after giving off the gastroduodenal artery, continues as the proper hepatic artery. The proper hepatic artery branches into the right and left hepatic artery and this branching is either intrahepatic or extrahepatic. The middle hepatic artery arises from either the right or left hepatic artery. Sometimes, there are multiple small middle hepatic arteries.

2.2.2 Portal supply

The portal vein carries splanchic blood to the liver and is formed by the confluence of the splenic vein and superior mesenteric vein. The confluence is extrahepatic. The portal vein divides into the right and left portal veins. The right portal branch divides into an anterior and a posterior branch and each of these divides into an ascending and a descending branch. The left portal branch also divides into an ascending and a descending branch. Figure 2.06 and figure 2.07 are schematic diagrams of the portal system.

2.2.3 Venous drainage

Generally the hepatic venous drainage consists of left, middle, and right hepatic veins which enter the inferior vena cava in the high subdiaphragmatic portion of the liver (figure 2.08)

Figure 2.05 Classical presentation of the arterial supply (from the coeliac axis) to the liver

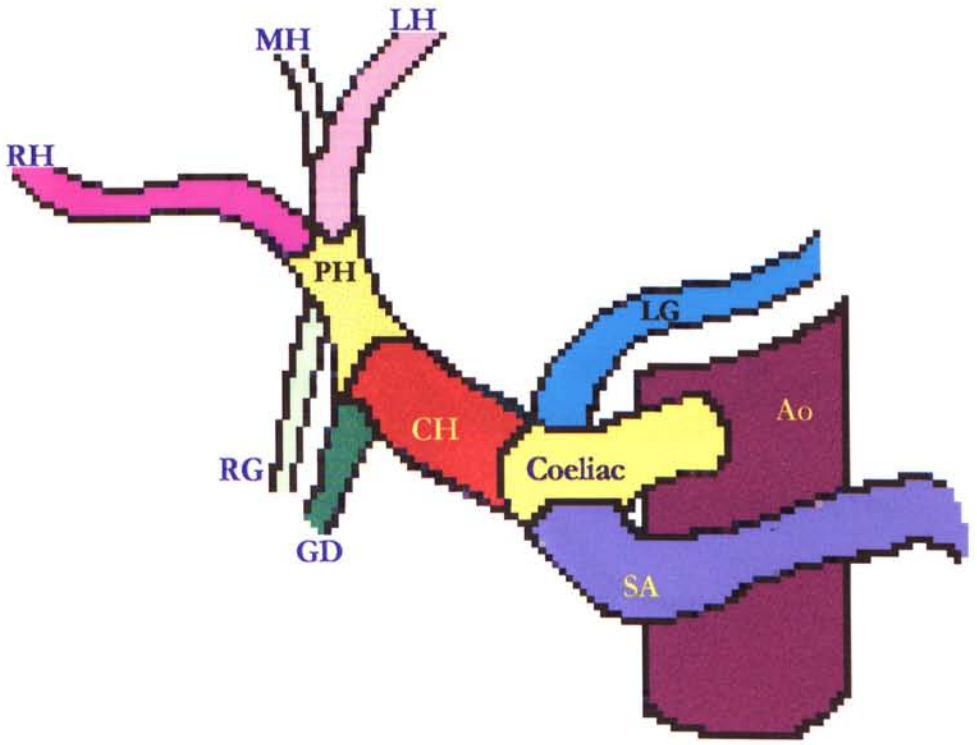


Figure 2.06 Schematic diagram of the portal veins.
(From Foundations of Anatomy and Physiology by Ross & Wilson)

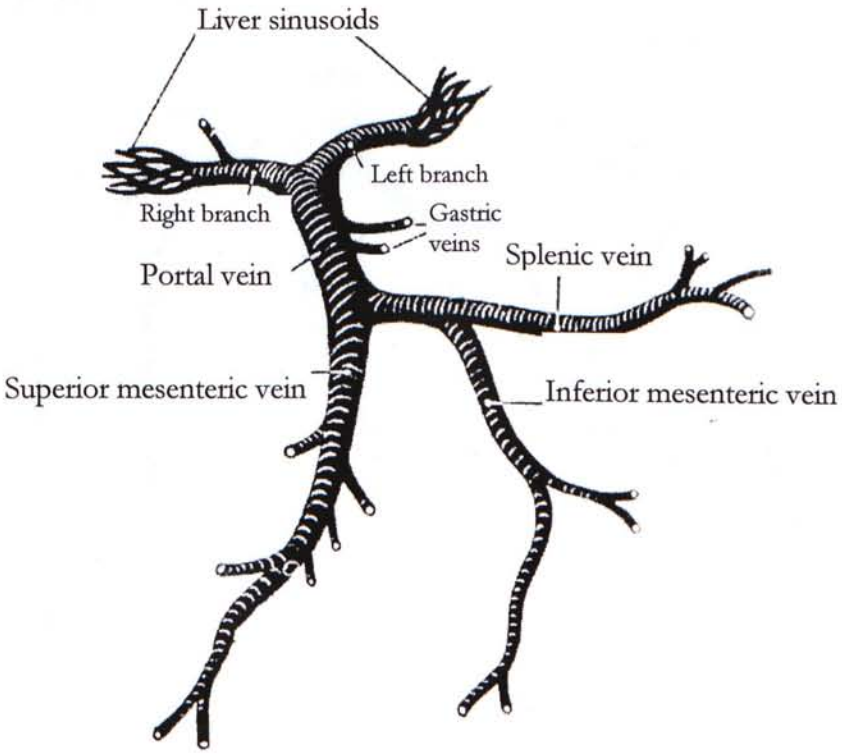


Figure 2.07 Schematic diagram of the portal veins in relation to the segments.
(From Modern Operative Techniques in Liver Surgery by Launois, Jamieson & Starzl)

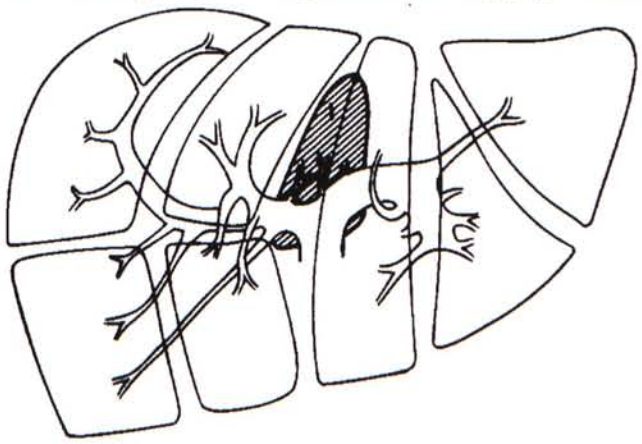
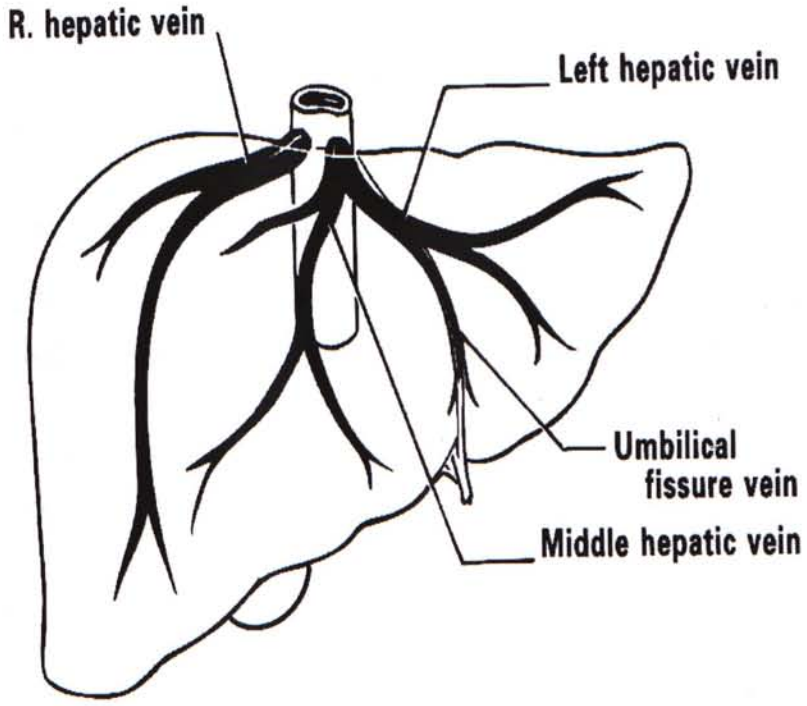


Figure 2.08 The major hepatic veins.
(From Modern Operative Techniques in Liver Surgery by Launois, Jamieson & Starzl)



2.3 Normal arterial anatomy of the coeliac axis

In this section, the 'normal' arterial anatomy of the coeliac axis is described. However, this 'normality' is only found in 65 to 75 per cent of all individuals, as cited from classical anatomy textbooks. Thus it is referred to as the classical presentation.

2.3.1 Development of the abdominal aorta:

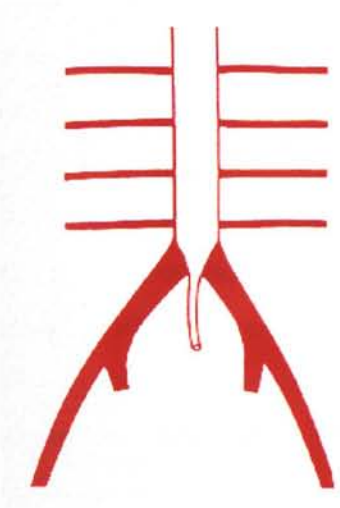
The branches of the abdominal aorta are divided into three groups: dorsal, lateral and ventral (figure 2.09).

The dorsal branches are segmental. During development, the lumbar arteries I - IV remain segmental arteries while the 5th artery forms the common iliac artery.

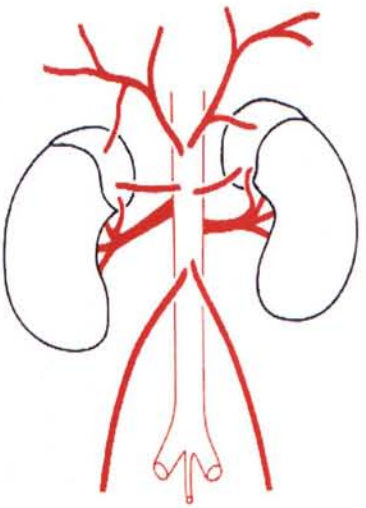
The lateral branches supply the kidneys and the genital organs.

The ventral branches develop initially as paired vessels. The arteries to the gut merge early to form the coeliac axis, superior mesenteric and inferior mesenteric arteries. The umbilical arteries are also ventral branches and longitudinal anastomoses with L5 artery develop. The initial origins from the aorta disappear. After birth, their distal parts form the lateral umbilical ligaments.

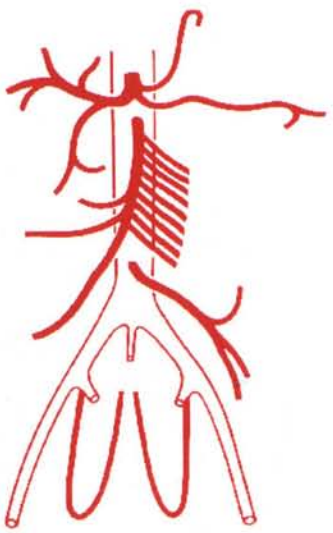
Figure 2.09 Branches of the abdominal aorta
(From Arterial variations in man. Classification and frequency by Munchen)



Dorsal



Lateral



Ventral

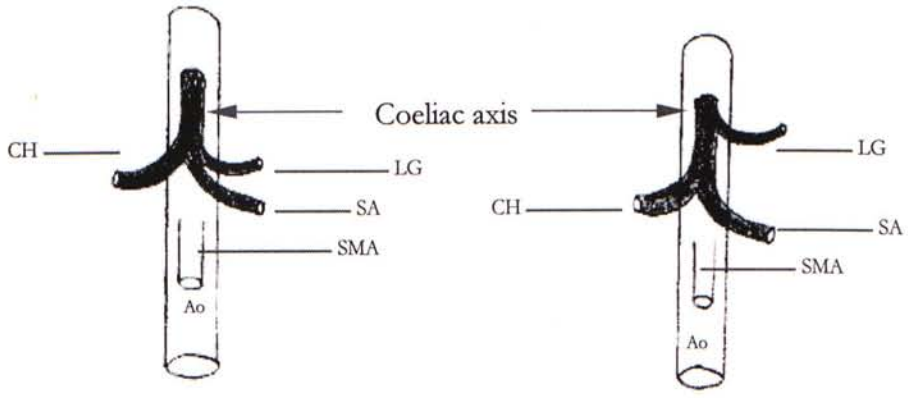
2.3.2 Coeliac axis

The coeliac axis arises anteriorly from the aorta between the twelfth thoracic vertebral body and the first lumbar vertebral body. The origin is usually slightly to the left of the mid line. The classical pattern is that of the 'complete coeliac axis' and its main branches include the gastric artery, the common hepatic artery and the splenic artery . The true trifurcation type (figure 2.10) accounts for only 25 per cent of all individuals. Another 'textbook' pattern (figure 2.11) accounts for 49 per cent of all individuals in which the coeliac axis gives rise to the left gastric artery as a side branch, and then divides more distally into the common hepatic artery and the splenic artery. (Lippert & Pabst, 1985)

Figure 2.10 Complete coeliac axis - true trification (25%)

Figure 2.11 Complete coeliac axis - LG as side branch of coeliac axis (49%)

(From Arterial variations in man. Classification and frequency by Lippert & Pabst*)*



2.3.3 Coeliac artery

The coeliac artery is the first portion of the coeliac axis. It averages 1 to 2 centimeters in length.

2.3.4 Left gastric artery

The left gastric artery arises from the coeliac axis in about 90 per cent of the population. It is commonly the first major branch of the coeliac axis (figure 2.11). It is the smallest of the three major branches. It usually courses along the lesser curvature of the stomach to anastomose with the right gastric artery.

2.3.5 Common hepatic artery

The common hepatic artery is one of the major branches of the coeliac axis. Normally it is slightly smaller than the splenic artery and it courses to the right. After giving off the gastroduodenal artery, the hepatic trunk is refered to as the proper hepatic artery. The proper hepatic artery then divides into the right and left hepatic arteries. The middle hepatic artery may arise from the right or left hepatic artery. This classical pattern constitutes about 50 per cent of the population (figure 2.12).

The gastroduodenal artery almost always arises before the common hepatic divides into the hepatic branches (figure 2.12).

The right gastric artery arises from the proper hepatic artery in 50 per cent of the population (figure 2.13). (Lippert & Pabst, 1985)

2.3.6 Splenic artery

The splenic artery arises from the coeliac axis and courses to the left of the body (figure 2.10, figure 2.11). It is usually the largest and the most tortuous branch of the coeliac axis.

Figure 2.12 Arterial blood supply of the liver from coeliac axis only
- CH is a branch of the coeliac axis (50%)
(From Arterial variations in man. Classification and frequency by Lippert & Pabst)

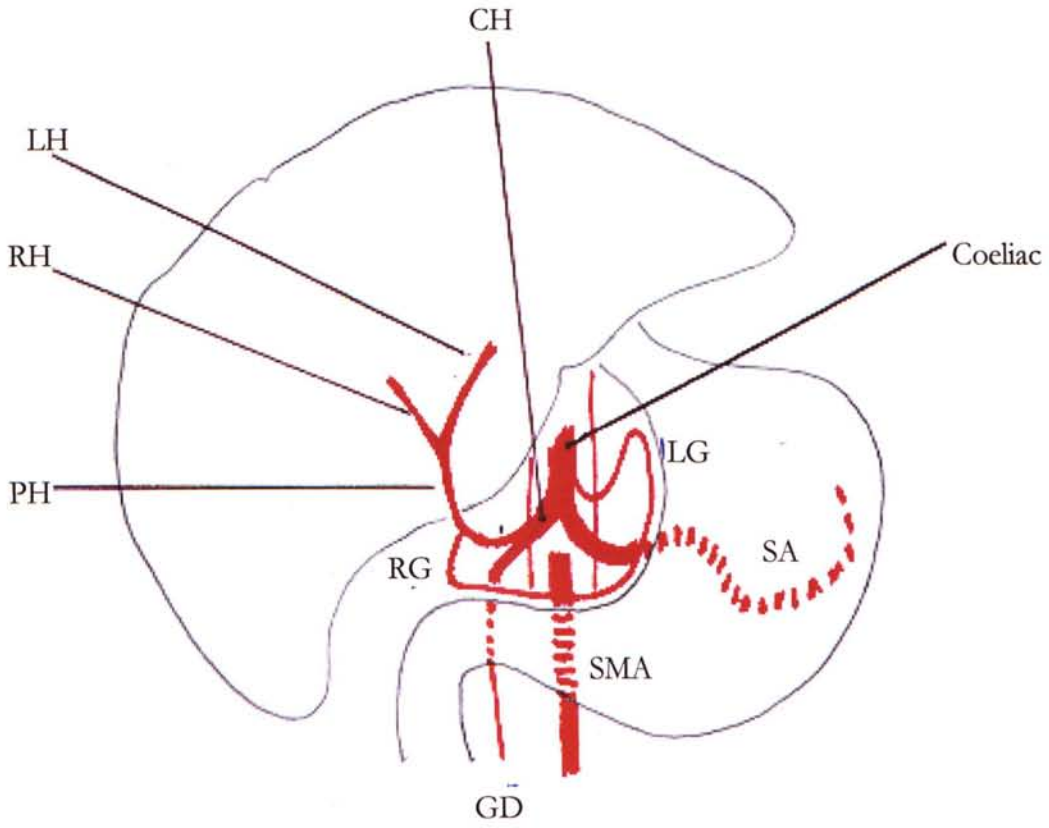
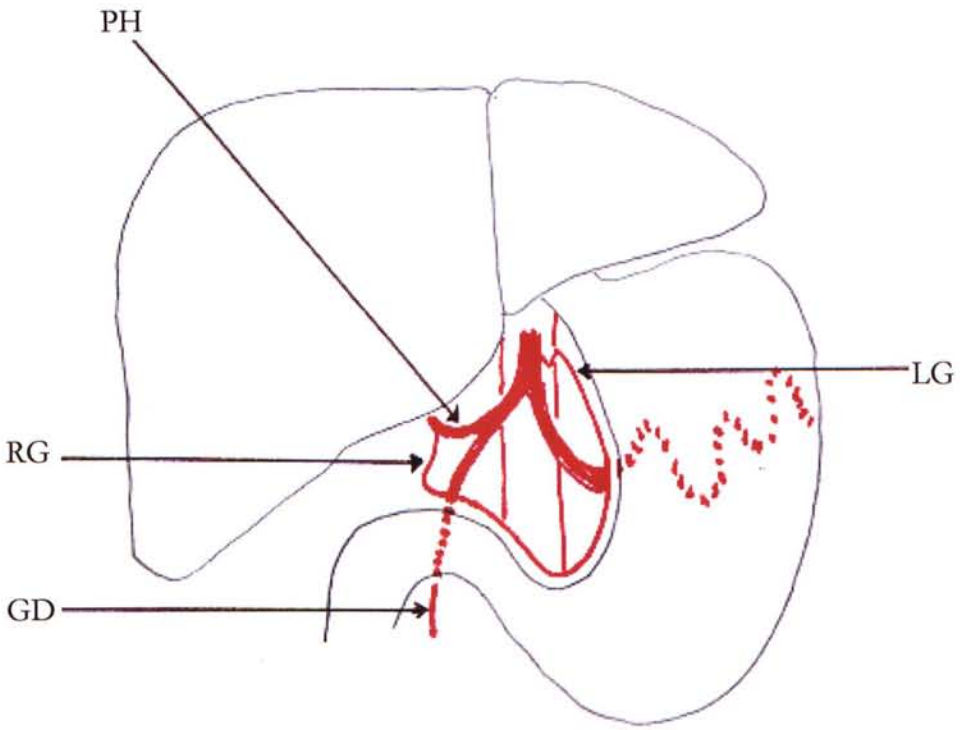


Figure 2.13 'Normal' origin of the right gastric artery - from PH (50%)
(From Arterial variations in man. Classification and frequency by Lippert & Pabst)



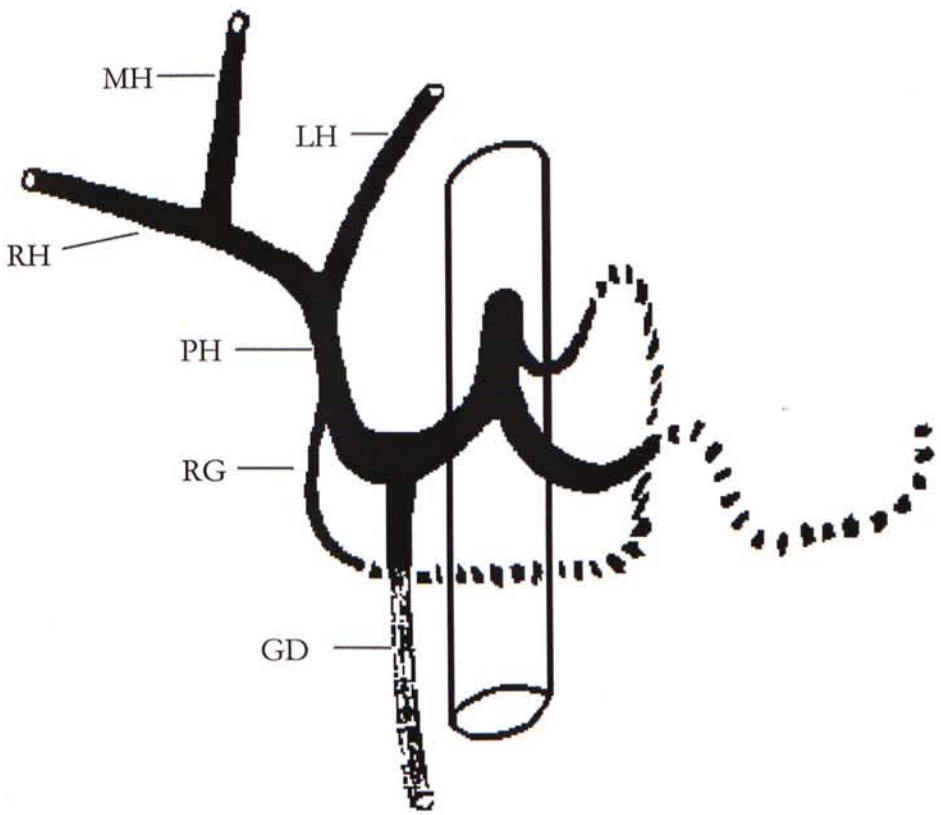
2.4 Common variations of the coeliac axis

2.4.1 Hepatic arterial supply

In about 55% of the population, the 'classical' hepatic arterial branching pattern is seen with the entire right, left, and middle hepatic arteries originating from the common hepatic artery (Saadoon, 1991). The proper hepatic artery is a continuation of the common hepatic artery and it divides into the right and left hepatic arteries. The middle hepatic artery may arise from the right or left hepatic artery. (Figure 2.14)

Other vessels in close relation with the hepatic arterial supply are the gastroduodenal artery and the right gastric artery. The gastroduodenal artery originates from the common hepatic artery in about 75% of the population and arises almost always prior to the division of the proper hepatic artery into right and left branches (Saadoon, 1991). (Figure 2.14) The right gastric artery branches from the proper hepatic artery in 40% of the population (Saadoon, 1991). (Figure 2.14) Both these vessels supply the upper gastro-intestinal tract and the pancreas. Their origins are especially important when performing endovascular intervention procedure of the liver

Figure 2.14 Hepatic arterial supply from the proper hepatic artery.
(From Arterial variations in man. Classification and frequency by Lippert & Pabst)



2.4.2 Normal variations of the hepatic arterial supply

Michels (1955) has pointed out that forty to fifty percent of individuals have significant anatomic variations in the coeliac and superior mesenteric arteries. These variations occur most often at the origins of the hepatic artery. The common variations of the coeliac axis and superior mesenteric artery can be explained by persistence of some or all of the primitive ventral anastomoses of the vitelline arteries and associated variations in the preservation of the tenth and thirteenth vitelline roots (Reuter, Redmann & Cho, 1986).(Figure 2.15, figure 2.16)

In understanding arterial variations one has to bear in mind the concept that all variations are due to preferential flow in normal arterial structures. During development, some arterial pathways develop and some involute. Embryologically, the liver bud derives from the duodenum. As the liver develops, the hepatic artery flow increases. The liver can then be considered as a collateral of the gastroduodenal artery. Because of the presence of a marginal vessel parallel to the digestive tube between the inferior diaphragmatic, left gastric, gastroduodenal, and superior mesenteric arteries, it is possible that the hepatic artery can haemodynamically derive from any of these vessels (figure 2.17). Table 2.01 lists some of the common variations which are important to the endovascular treatment of hepatic lesions.

(see Appendix II for embryology)

Figure 2.15 The primitive vascular supply - All segmental arteries and a ventral anastomosis are present

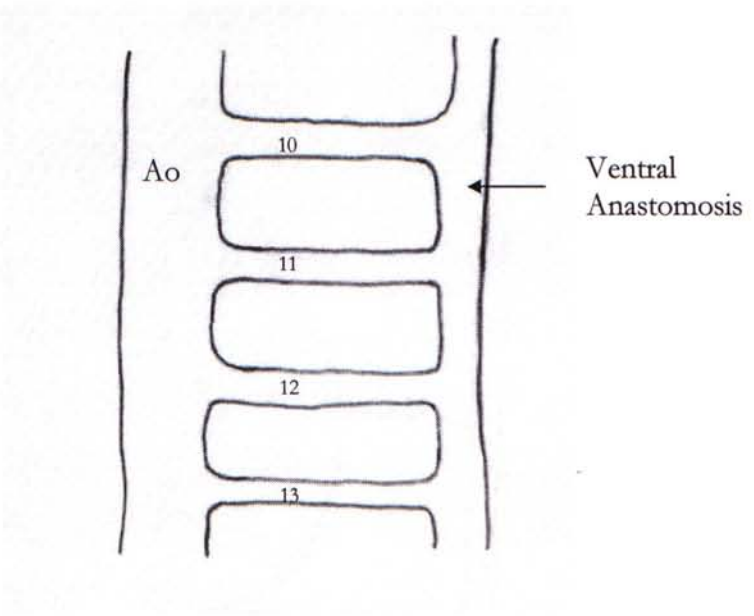


Figure 2.16 The 10th root forms the coeliac axis, the 13th forms the SMA, the 11th & 12th root and the ventral anastomosis regress

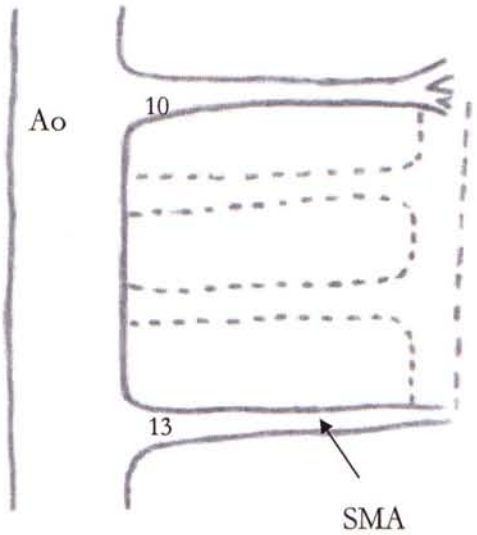


Figure 2.17 Schematic showing vascularising state of the digestive tube embryologically

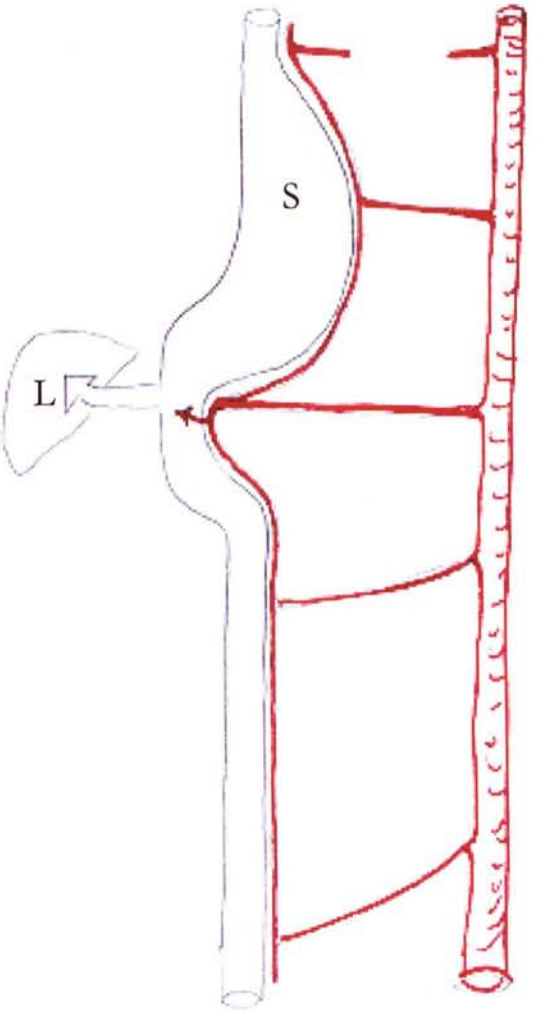
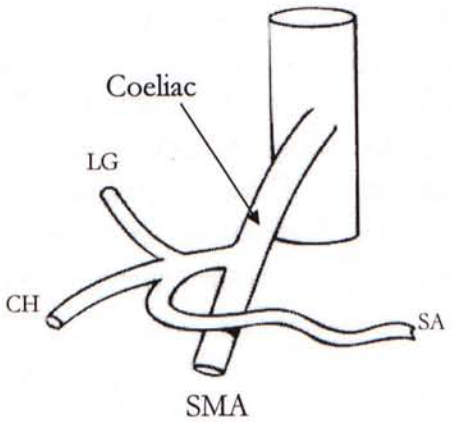


Table 2.01 Variant anatomy of the hepatic arterial supply

Type	Incidence	Illustration
Coeliacomesenteric trunk (Coeliac axis and superior mesenteric artery have same origin.)	< 1%	Figure 2.18
Common hepatic artery from aorta	2%	Figure 2.19
Common hepatic artery from superior mesenteric artery	2.5%	Figure 2.20
Right hepatic artery from superior mesenteric artery Replaced right hepatic Accessory right hepatic	14-18% 10-12% 4- 6%	 Figure 2.21 Figure 2.22
Left hepatic artery from the left gastric artery	12%	
Accessory left hepatic from the left gastric artery	6%	Figure 2.23

Figure 2.18 Coeliacomesenteric trunk *Figure 2.19 CH from Aorta*
(From Modern Operative Techniques in Liver Surgery by Launois, Jamieson & Starzl)



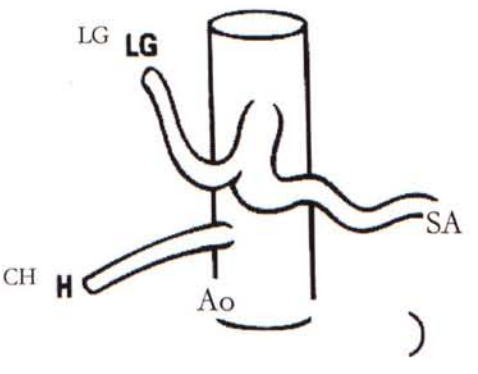
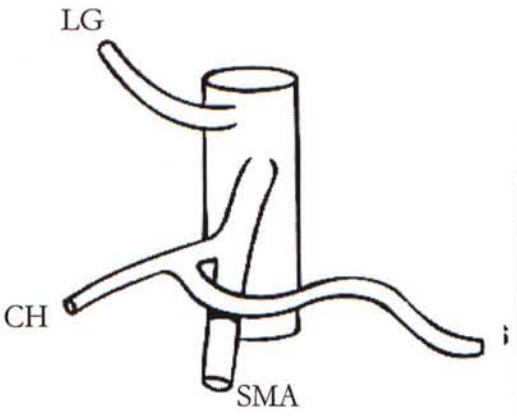


Figure 2.20 CH from SMA *Figure 2.21 RH from SMA*
(From Modern Operative Techniques in Liver Surgery by Launois, Jamieson & Starzl)



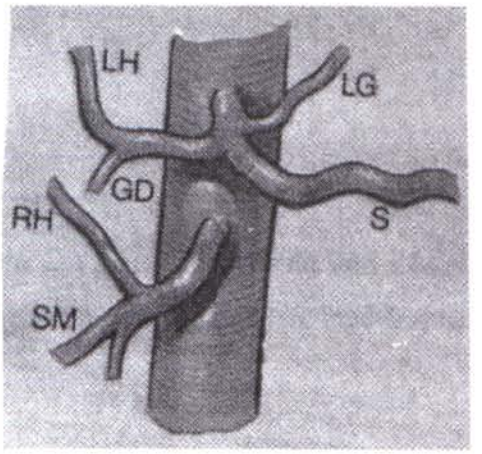
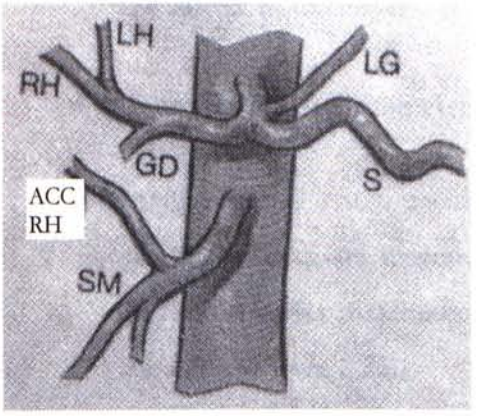
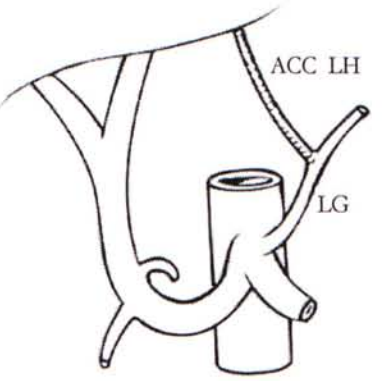


Figure 2.22 ACC RH from SMA *Figure 2.23 ACC LH from LG*
(From Modern Operative Techniques in Liver Surgery by Launois, Jamieson & Starzl)





2.5 Previous classification of coeliac anomalies

In this section, Michels' classification is described.

2.51 Types of coeliac axis

- Type I. Hepatolienogastric Trunk

  This type of coeliac trunk has the left gastric, the splenic and a hepatic artery - either the common hepatic or its right or left branch.(Figure 2.24)

- Type II. Hepatolienal Trunk

  In this type, the hepatic and the splenic arise from a common coeliac trunk. The left gastric is displaced, i.e., arises separately from the aorta, the splenic or the hepatic. (Figure 2.25)

- Type III. Hepatolienomesenteric Trunk

  In this type, the sole coeliac component remaining in situ is the left gastric, which arises separately from the aorta. The hepatic, the splenic and the superior mesenteric arise from a common trunk derived from the aorta. (Figure 2.26)

- Type IV. Hepatogastric Trunk

  In this type, the left gastric and the hepatic arise from a common trunk at the coeliac site and the splenic arises from the superior mesenteric. (Figure 2.27)

- Type V. Lienogastric Trunk

  In this type of coeliac trunk, the splenic and the left gastric arise from a common trunk, and the hepatic artery is replaced from another source. (Figure 2.28)

- Type VI. Coeliacomesenteric Trunk

  Here, the four regional arteries (hepatic, left gastric, splenic and superior mesenteric) arise from the aorta by a common trunk.

- Type VII. Coeliacocolic Trunk

  In this type, the middle colic or the left colic takes origin from the coeliac instead of from the superior mesenteric. (Figure 2.29)

  (See Appendix III for the occurrence percentages of the different types of coeliac axis, by Michels' study)

Figure 2.24 Hepatolienogastric Trunk
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)

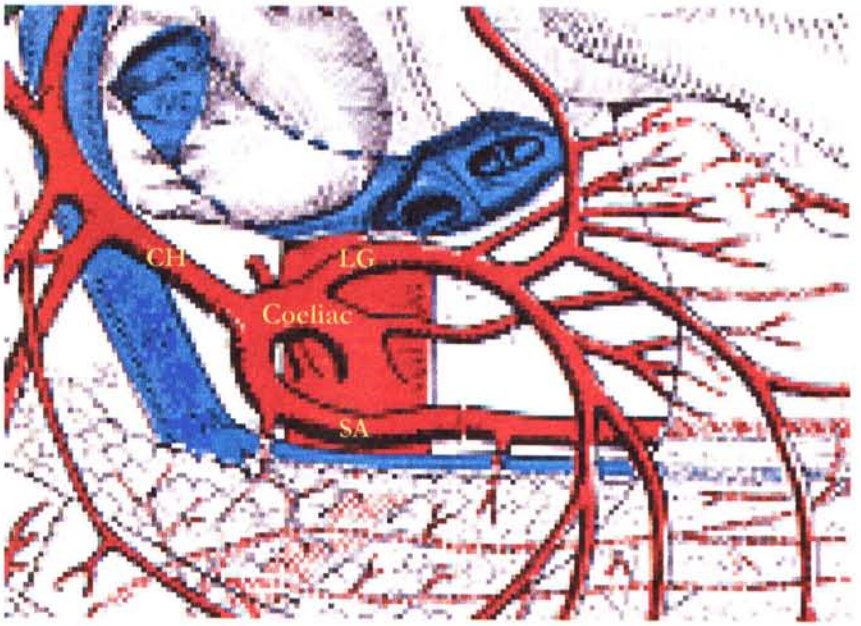


Figure 2.25 Hepatolienal Trunk
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)

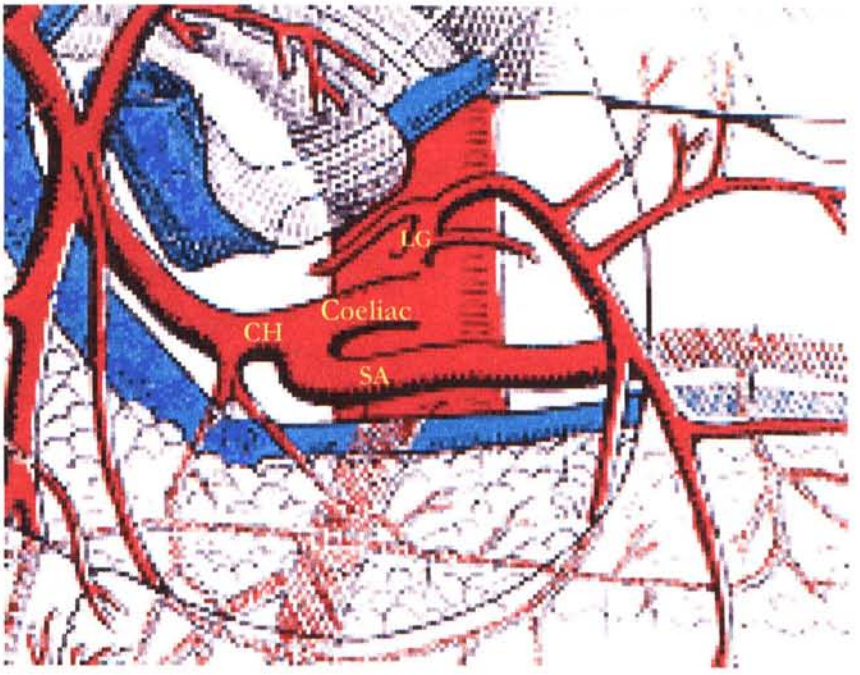


Figure 2.26 The hepatic, the splenic and the SMA arise from a common trunk from the aorta
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)

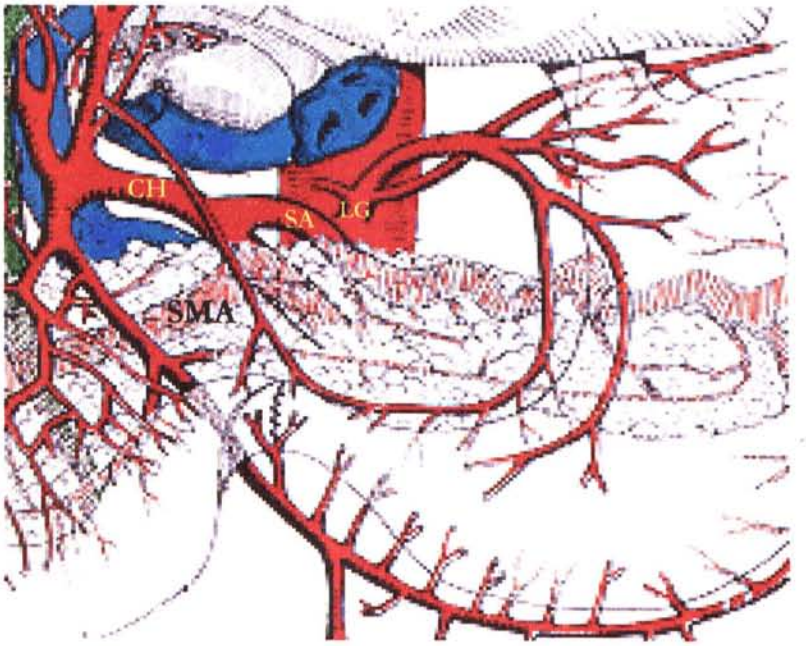


Figure 2.27 The left gastric and the hepatic from a common trunk at the coeliac, SA fr SMA
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)

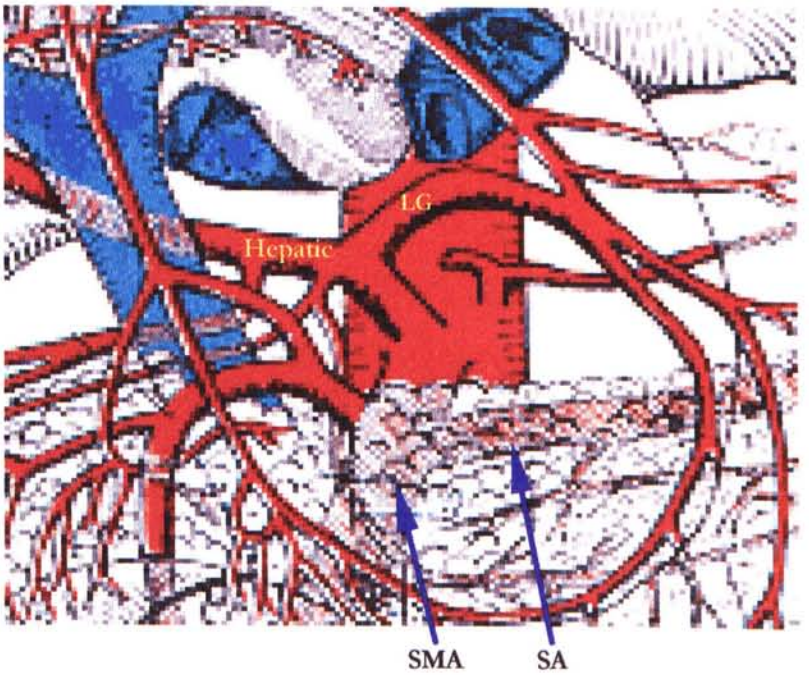


Figure 2.28 The splenic and the left gastric arise from a common trunk at the coeliac, the hepatic is replaced from other source
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)

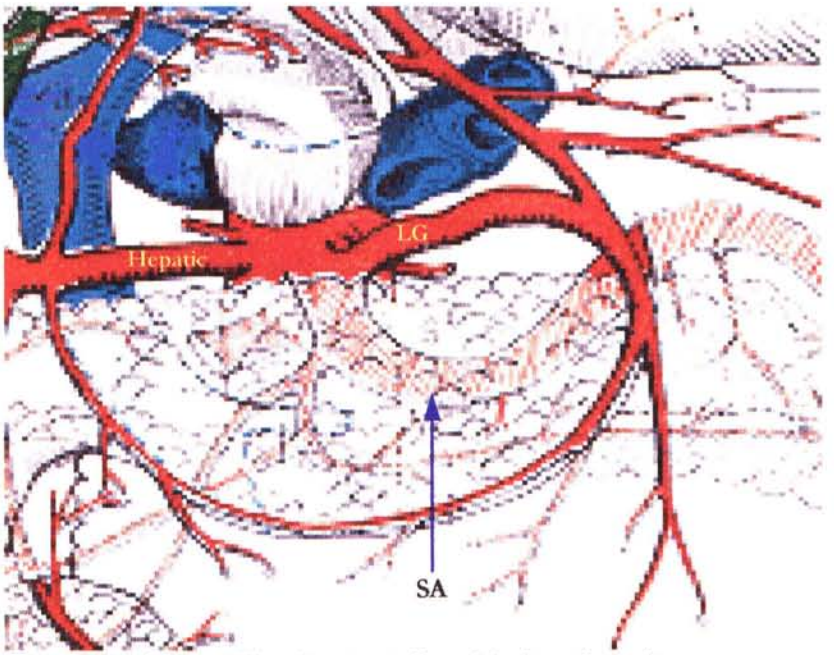
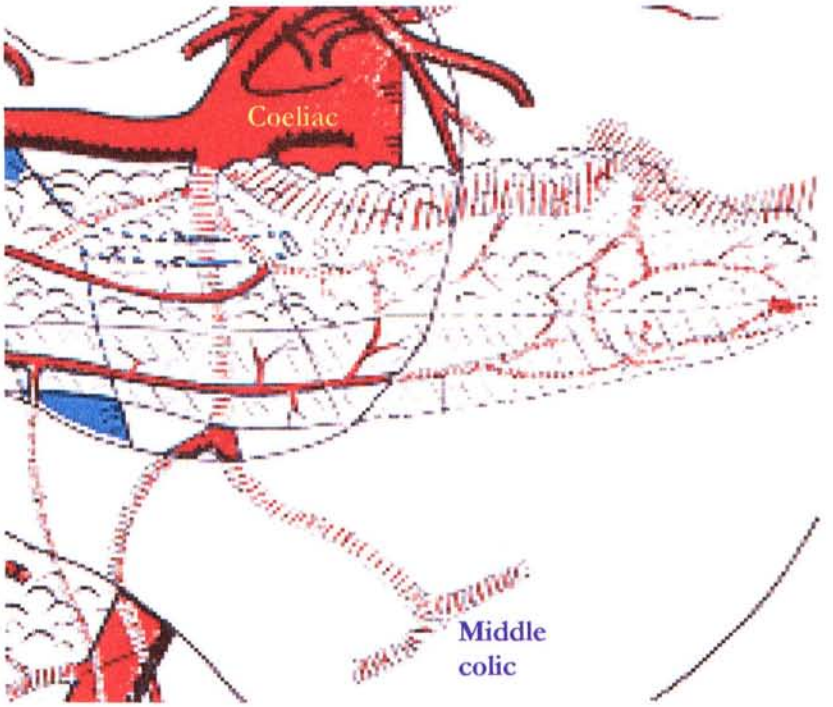


Figure 2.29 The middle colic artery takes origin from the coeliac
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)



2.52 Basic types of the hepatic arterial blood supply

- Type I.

  This is the textbook type, where the coeliacal hepatic supplies the right, the left and the middle hepatics.

- Type II

  The coeliacal hepatic supplies the right and the middle hepatics and the left hepatic replaced from the left gastric.

- Type III

  The coeliacal hepatic supplies the left and the middle hepatics while the right hepatic is replaced from the superior mesenteric.

- Type IV

  The coeliacal hepatic supplies the middle hepatic only. The right hepatic is replaced from the coeliac or the superior mesenteric, and the left hepatic is replaced from the left gastric.

- Type V

  The coeliacal hepatic supplies the right, the left and the middle hepatics. There is an accessory left hepatic from the left gastric

- Type VI

  The coeliacal hepatic supplies the right, the left and the middle hepatics. There is an accessory right hepatic from the superior mesenteric.

- Type VII

  The coeliacal hepatic supplies the right, the left and the middle hepatics. In addition, there is an accessory right hepatic from the superior mesenteric and an accessory left hepatic from the left gastric.

- Type VIII

  This type is a combination of two patterns:

  (i) There is a replaced right hepatic (i.e. not from the coeliacal hepatic trunk) and an accessory left hepatic.

  (ii) There is a replaced left hepatic (i.e. not from the coeliacal hepatic trunk) and an accessory right hepatic.

- Type IX

  The entire hepatic trunk is derived from the superior mesenteric with the coeliacal hepatic absent.

- Type X

  The entire hepatic trunk is derived from the left gastric when the coeliacal hepatic is absent.

  (See Appendix IV for the occurrence percentages of the different types of hepatic arterial supply, by Michels' study)

2.6 Knowledge of arterial anomaly in relation to surgery

2.6.1 Preparation for liver resection

The surgeon must answer three important questions when evaluating a patient for liver surgery. The questions are :

1. Can the lesion be removed completely without damage to vital structures?

2. Will the patient's general health allow the patient to survive a major operation?

3. Does the patient possess enough liver reserve and capacity for liver regeneration to permit removal of the amount of liver tissue required to eliminate the disease?

In many cases, arteriography, venography, and cholangiography can aid in the evaluation of anatomic resectability. In the context of this study, we are interested in the arteriography and the information it provides, in relation to liver surgery, about the vascular map and any anomaly present in the patient.

2.6.2 Attention with regard to hepatic arteries

For liver resection, attention with regard to hepatic arteries are :

- At the hilum, after opening the Calot's triangle, while palpating the hepatoduodenal ligament for any portal nodal spread of tumor it is necessary to make sure whether there are any aberrant right hepatic arteries arising from the superior mesenteric artery. (R. Scott Jones, 1990)

- During hepatic artery ligation in right hepatectomy, the right hepatic artery should be preserved as long as possible and tied extremely close to the parenchyma, because vascular supply of the common hepatic duct is mainly derived from the right hepatic artery. (R. Scott Jones, 1990)

- Accessory left hepatic artery may arise from the left gastric artery. Ligation of this vessel is required in left hepatectomy. (R. Scott Jones, 1990)

- For liver trauma, selective ligation of the hepatic artery is indicated when intrahepatic ligation or suture has failed to control hepatic arterial haemorrhage. (Launois, Jamieson, and Starzl, 1993)

- During hepatic lobectomy the surgical team must avoid injury of the vessels and ducts to the lobe that must remain. (R. Scott Jones, 1990)

2.7 Trans-catheter treatment of HCC

2.7.1 Treatment of HCC

Although surgery is the preferred treatment for HCC, a mortality rate as high as 33% is associated with certain hepatic resections. (Melvin E. Clouse, 1983)

2.7.1.1 Intravenous chemotherapy

In the early eighties, intravenous chemotherapy had been attempted. The drugs used included Adriamycin (ADM, doxorubicin), 5-fluorouracil (5-FU) and Methyl-CCNU (MeCCNU). These drugs were used either as a single agent therapy or combination drug programs, but these attempts to palliate hepatoma patients had generally resulted in more frustration than benefit. Furthermore, when the toxicity of treatment is borne in mind it is clear that none of the intravenous chemo-treatments can be recommended as a standard treatment approach. (G. Falkson & J.M. MacINTYRE et al, 1984)

2.7.1.2 Conventional radiotherapy

Conventional radiotherapy was of " no benefit " for patients with HCC. This conclusion was based on the use of 2,000 to 3,000 rad as a single modality. Hepatoma is too virulent a disease for this modest radiation dose to be effective. However, the use of larger dose/fraction external radiation therapy is an effective means of symptom palliation. From M. A. Friedman's study, data showed that doses of 2,500 to 3,500 rad could be tolerated without undue risk of radiation hepatitis, but studies also showed that external radiation alone provided only temporary, modest benefits, at best. (Michael A. Friedman, 1983)

2.7.1.3 Internal irradiation with radioimmunoglobulins

Internal irradiation with radioimmunoglobulins is another form of radiotherapy for HCC. With the development of various monoclonal antibodies such as antiferritin and anti-AFP, which are to some degree tumour specific, HCC

can be targeted after systemic administration of the appropriate antibodies. By labelling the antibodies with iodine-131 or yttrium-90, a therapeutic dose of radiation can be delivered to the tumour specifically. The treatment was preceded by whole liver external beam irradiation, and radiosensitizers (doxorubicin and 5-Fu) were added in the latter phase of the trial. The treatment was compared with systemic chemotherapy in a randomised trial which showed comparable response rates and survival duration. Those failing chemotherapy could still respond to ^{131}I-antiferritin treatment. Though the treatment is given systematically, its effect is mainly locoregional and the dose of^{131}I-antiferritin is not sufficient to treat extrahepatic disease on its own. This is more effective than external radiotherapy alone. (P.J.Johnson, S. Ho and W.T.Leung, 1994)

The above described was before the era of regional targeted treatment for HCC. In the early nineties, selective internal radiation has gained ground as the preferred treatment of this "difficult" tumour.

2.7.2 Trans-catheter treatment of HCC

Before the nineteen nineties there was no standard trans-catheter therapy for HCC patients. The trend was towards using intra-arterial regional targeted treatment since it was quite definite that malignant neoplasms growing in the liver had an exclusively arterial blood supply (Breedis and Young, 1954). It had been suggested that liver transplantation, if successful, might be the ideal treatment for cirrhosis-based HCC. However, this was costly and its contribution was very limited on a global scale (Okuda K. et al, 1985).

The types of trans-catheter treatment of HCC includes :

2.7.2.1 Intra-arterial infusion chemotherapy

Both primary and metastatic hepatic neoplasms derive their blood supply and nutrition from the arterial circuit. Thus the hepatic artery is an ideal route for selective delivery of therapeutic agents. The stimulus for using infusion therapy arises because of a poor median survival time in untreated patients and those receiving systemic chemotherapy. By infusing drugs directly into the tumour via the hepatic artery, local concentration of the anti-tumour agent is increased and systemic toxicity is decreased. Successful arterial infusion therapy depends on adequate perfusion of the entire liver and a safe delivery system. In early studies, a catheter was inserted percutaneously via the brachial or femoral artery but this approach suffered from the disadvantages that local and systemic sepsis was common after protracted infusions and that the patient was confined to bed and hospital. To overcome these problems, totally implantable systems have been developed. These systems are not without disadvantage. The implanted pumps are very expensive and require laparotomy for their insertion; those acting as a port are not easy to keep patent and in place over several months. The infusion of chemotherapy may result in chemical hepatitis or gastritis and ulceration. One major problem with this approach in HCC is the lack of effective drugs.

2.7.2.2 Hepatic artery embolization

Hepatic artery embolization creates tumour devascularization, but the portal flow prevents infarction of liver parenchyma because of the single vascular supply from the hepatic artery to a neoplasm, in contrast to the dual vascular supply to the liver parenchyma. In the early eighties, indications for the use of hepatic artery embolization are failure of chemotherapy, either systemic or intra-arterial infusion, vascular anomalies requiring combined lobar embolization and lobar infusion, and lack of effective treatment

Mainly there are three types of embolization : peripheral embolization using Gelfoam, proximal embolization using coils, and combined peripheral and proximal embolization. The median survival duration was reported to be 11.5 months from the time of embolization.

Intrahepatic collaterals have been observed following both surgical ligation and coil embolization of the hepatic artery; some of these collaterals open instantaneously, others develop with time. To minimize collateral circulation and to create a more completely ischemic tumour, peripheral embolization of the hepatic artery is undertaken. Repeat embolization is necessary weeks to months later, because angiograms usually reveal inconsistent occlusion, probably due to resorption of Gelfoam and recanalization of hepatic arteries. Thus, proximal embolization of the hepatic artery following peripheral embolization with Gelfoam was the usual practice in the early eighties in North America. (V.P.Chuang and S.Wallace, 1981)

Now, in the nineties, simple arterial embolization on its own is less used in HCC, and this technique is usually used in combination with chemotherapy and other methods of loco-regional treatment. Its value in controlling severe pain has been recognised and it is still used for slow growing metastatic hormone-secreting hepatic tumours where it can lead to symptomatic control for several years. (P.J.Johnson, S. Ho and W.T.Leung, 1994)

2.7.2.3 Transcatheter oily chemoembolization (TOCE)

Lipiodol, when injected into the hepatic artery at the time of arteriography accumulates in hepatocellular carcinoma. Several groups from the Far East have suggested that Lipiodol might be an ideal vehicle for targeting cytotoxic drugs because, after mixing with a therapeutic agent, it leads to an increased concentration of drug in the tumour. (P.J.Johnson, S. Ho and W.T.Leung, 1994)

Most commonly 60mg of doxorubicin is thoroughly mixed with 5ml of Lipiodol and injected into the tumor feeding arteries. This is followed by embolization with Gelfoam particles. It is important to confirm before the procedure that the portal vein is patent and, after the procedure, that the appropriate arteries have been embolized. The treatment is repeated on several occasions as the Gelfoam is reabsorbed within a few weeks and there is rapid recanalization. (P.J.Johnson, S. Ho and W.T.Leung, 1994)

Large series have been reported from Japan with survival figures at one year of around 50-75%, similar to those obtained by surgery. By the late eighties, the procedure was widely regarded as standard treatment. Later, studies showed that TOCE caused reduction in tumour size but there was little effect on long-term survival. Furthermore, most of the effect seen is due to the embolization, there being no proof that the Lipiodol targeting adds significantly. (P.J.Johnson, S. Ho and W.T.Leung, 1994)

2.7.2.4 Selective internal radiation with Lipiodol-iodine-131

HCC has an avidity for Lipiodol (Hoogewoud, 1993). By giving the radioactive Lipiodol-iodine-131 intra-arterially, a therapeutic dose of irradiation can be delivered to the tumour. This treatment method has the theoretical advantage of preferential uptake of the radioactive iodine by the HCC and possibly decreases systemic toxicity. The thyroid is blocked by non-radioactive iodine before treatment to prevent uptake of the radioisotope. (P.J.Johnson, S. Ho and W.T.Leung, 1994)

The radioactive Lipiodol-iodine-131 is given slowly through an angiographic catheter which is placed in the tumor feeding hepatic artery. (P.J.Johnson, S. Ho and W.T.Leung, 1994) Since Lipiodol is radiopaque, the flow of the substance can be monitored under fluoroscopy.

Lipiodol-iodine-131 emits mainly gamma radiation with an energy of 364 keV. Due to this relatively low energy, its use is limited to tumours of less than 5 cm in diameter. The dose given ranges from 555 to 2220 MBq. (P.J.Johnson, S. Ho and W.T.Leung, 1994). The physical half-life of iodine-131 is 8.04 days. The biological half-life of the radioactive Lipiodol varies from patient to patient depending on blood flow, arteriovenous shunting and clearing by the reticuloendotheial cells. After treatment, the patient has to stay in hospital for approximately 10 to 14 days in order to allow the radiation to decay to a safe level before discharging from the hospital. (P.J.Johnson, S. Ho and W.T.Leung, 1994)

No major side effects were reported. (Bretagne and Raoul et al., 1988) Distribution of Lipiodol-iodine-131 can be detected by gamma camera and dosimetry data showed that HCC would receive on average eight times more irradiation than normal liver. (P.J.Johnson, S. Ho and W.T.Leung, 1994) However, from Hoogewoud, results seemed similar to those of other techniques such as chemoembolization. One of the advantages of internal radiation therapy is that it preserves hepatic vasculature and does not exclude the possibility of liver transplantation. . (Bretagne and Raoul et al., 1988) Despite the advantages, a limiting factor is the patient's psychological tolerance of isolation.

2.7.2.5 Selective internal radiation with yttrium-90 microspheres

The injection of radioactive microspheres through the hepatic artery is particularly appealing because it allows local administration of high radiation doses to the tumour without significant hepatic or systemic toxicity. (F.A. Shepherd & L.E. Rotstein, 1992) Yttrium-90 has several properties that make it a desirable agent for internal radiation therapy. It is a pure beta emitter with a mean energy of 0.93MeV (maximum, 2.27MeV). The average penetration in tissue is 2.5mm. The physical half-life is 64.2 hours. Stable ^{89}Y is incorporated easily into a glass matrix and can be activated to radioactive ^{90}Y by means of neutron bombardment in a nuclear reactor. (F.A. Shepherd & L.E. Rotstein *et al*, 1992)

^{90}Y microspheres are not biodegradable. Once infused into the liver, they will stay in the microvasculatures of the tumour. In the early phase, the treatment procedure required laporotomy. The problems encountered included:

- Myelosupression resulting from leaching of the radionuclide.
- Pulmonary fibrosis resulting from arteriovenous shunting in the tumour circulation.
- Gastritis due to the passage of radioactive microspheres into the gastroduodenal vessels.
- Inability to calculate the radiation dose administered to the liver.

The above problems were later solved by the followings :

- The invention of the new ^{90}Y glass microspheres. (M.J. Herba & F.F. Illecas et al, 1988)
- Tc-MAA scan to predict the percentage lung shunting.
- Tc-MAA scan to predict the relative T/N ratio.

From October of 1992, laparotomy for ^{90}Y microspheres therapy has no longer been performed since pre-treatment simulation using Tc-MAA scan to predict percentage of lung shunting and T/N ratio was verified. Now, ^{90}Y-SIR therapy is performed during HAG with pre-treatment Tc-MAA scan. (Leung Wai-Tong, 1995) Although the technique for injecting ^{90}Y microshperes is very much skill dependent and despite the large amount of activity handled during treatments, exposure of the personnel involved is minimal. (Hoogewoud, 1993)

2.7.2.5.1 Procedure of ^{90}Y microspheres administration

1. ^{90}Y microspheres are made radiopaque by mixing with an equal volume of radiopaque contrast medium, Omnipaque 300.

2. The suitable patient is subjected to HAG using the Seldinger technique.

3. Arteriograms of the coeliac artery, superior mesenteric artery and hepatic artery are performed and carefully studied for the vascular map with special attention to any anomalies present. This is to accurately assess the tip of the catheter placement for the ^{90}Y microspheres infusion.

4. Catheter tip is placed in the feeding artery of the tumour or the proper hepatic artery distal to the branching off of the right gastric artery.

5. Catheter position is the same as that for the previous pre-treatment HAG with Tc-MAA scan.

6. Catheter is then connected to a 4-way valve fixed on an injection bracket made with 1cm thick perspex.

7. A pre-determined dose of ^{90}Y microspheres basing on the T/N ratio from pre-treatment Tc-MAA scan, is given into the angiographic catheter.

8. The flow of the ^{90}Y microspheres is monitored closely under fluoroscopy.

9. The catheter is removed after the treatment and haemostasis secured.

10. After the treatment, the patients are nursed in an isolation ward for the purpose of radiation protection and discharged home on day five post

treatment when the radiation has decayed to a safe level. (Leung Wai-Tong, 1995)

2.8 Prevalence of HCC in Hong Kong Chinese

HCC is the most frequently occurring malignant tumour in the liver. It is currently the second commonest cancer death in Hong Kong. The incidence rate was 28.6 per 100,000 for the male and 7.7 per 100,000 for the female population in 1984. A total of 1,000 cases were registered in the year with a male to female ratio of 4 to 1. It consisted of 7.5% of all the cancer cases seen in Hong Kong. The incidence rate was rising steadily over the last 20 years from 16.9 in 1965 to 28.6 per 100,000 in 1984. (Shiu *et al.*, 1990)

HCC is the seventh most common form of cancer in males and the ninth in females in Hong Kong. HCC accounts for 8.5% of all newly diagnosed cases of malignancy and 12.5% of all cancer deaths (Hong Kong Cancer Registry 1989, March 1993). In the year 1993, the number of deaths from malignant neoplasm of liver was 1168. The number of deaths has been increasing from 992 in 1984 to 1168 in 1993 (annual report of the Department of Health of Hong Kong, 1st April 1993 - 31st March 1994). (Appendix V)

2.9 Management of HCC in Hong Kong

In the Prince of Wales Hospital of the Chinese University of Hong Kong, the Joint Hepatoma Clinic takes care of the HCC patients. Previous study showed that, at presentation 83% of HCC patients had evidence of hepatomegaly suggesting that most of the tumours were very advanced and often beyond cure. The operability rate was 27% after ultrasonographic examination and this percentage dropped to 17% after hepatic arteriographic examination (W.T. Leung etal, 1994). The usual practice of the Joint Hepatoma Clinic is that all new patients are referred for ultrasound examination. After USG examination, the potentially operable patients are scheduled for HAG examination to further confirm operability. The operable patients are prepared for definitive surgery with abdominal CT scan and other pre-operative preparations. Those patients with bilobe disease, and/or extra-hepatic metastasis and/or portal vein invasion are considered inoperable. The inoperable patients are considered for palliative treatments. Palliative treatments include systemic chemotherapy, regional targeted treatment and general supportive care. (See Appendix VI for flow chart for HCC management) The vascular anatomy of each individual has great influence on the actual regional targeted treatment procedure. Thus, arterial variations are one of the important influencing factors upon the success of regional targeted treatment.

Chapter 3

DEFINITIONS

Considerable confusion often arises as regards the nomenclature of the arteries emanating from the coeliac axis. According to Michels, 1955, variational anatomy of the coeliac artery is so extensive that, in an investigation of 200 bodies, only about one half of the samples will conform to the standard anatomy textbook description. The main cause of aberrant morphology of the coeliac axis is the frequency and the diversity of origin and distribution of the hepatic arteries (Michels, 1955). It is suggested that the term coeliac axis be referred to the stem which typically gives rise to the left gastric, the splenic and the hepatic artery which then branches into the right, middle and left hepatic arteries.

In this study, the researcher concentrates on the origins of the vessels and the hepatic arterial supply is considered the core. Basically, the suggested terminology is followed with the coeliac axis defined to be the stem which gives rise to the left gastric artery, and/or the splenic artery, and/or the hepatic arteries. Thus, the 'normal' left gastric artery, the 'normal' splenic artery and the 'normal' common hepatic artery are all defined to be arising from the coeliac axis.

3.1 Definition of normality

With reference to the 'anatomy textbook normal' presentation of the coeliac axis and its branches, the definitions are:

- Coeliac axis from aorta
- Left gastric artery from coeliac axis
- Splenic artery from coeliac axis
- Common hepatic artery from coeliac axis
- Proper hepatic artery from common hepatic artery
- Right hepatic artery from proper hepatic artery
- Left hepatic artery from proper hepatic artery
- Middle hepatic artery from right hepatic artery or left hepatic artery
- Gastroduodenal artery from common hepatic artery
- Right gastric artery from proper hepatic artery

3.2 Accessory right or left hepatic artery

With regards to the accessory hepatic arteries, the term 'accessory' only applies when there are more than one right or left hepatic arteries. The more distal artery or the one which is not from the coeliac axis is termed the accessory right or accessory left hepatic artery. Here the term accessory implies an 'extra' vessel. This 'extra' is to be interpreted only from the viewpoint of origin, for, functionally considered, all hepatic arteries are essential, as proved in the plastic casts made by Healey and Schroy in 1952 (Michels, 1955).

3.3 Middle hepatic artery

The middle hepatic artery is not described or illustrated in anatomy texts. Michels said that the middle branch of the hepatic artery was known since the time of Haller (1756) and of Tiedemann (1822) and it should be included in all anatomy texts. Healey and Schroy designated the artery as the medial segment artery, for, they found that the middle hepatic (medial segment artery) supplied the medial segment of the left lobe of the liver. In Michels' study, about 45% of the middle hepatic arose from the right hepatic, about 45% from the left hepatic and 10% from other sources. Thus, whether the middle hepatic arises from the right or the left hepatic, it is considered a 'normal' presentation.

Chapter 4

OBJECTIVES

The aim of this study is to study the 'normal' and variant hepatic arterial supply and how the variant vascular pattern influence the trans-catheter ^{90}Y-SIR treatment for HCC. The objectives are five folds :

1. To establish the percentages of the variant origins of the coeliac axis and its branches in Hong Kong Chinese.

2. To provide illustrative arteriograms of the observed anomalies.

3. To find the influence of the anomalies upon trans-catheter ^{90}Y-SIR therapy procedure.

4. To confirm that the presence of anomalies is not a pre-disposing factor for HCC.

5. To confirm that there is no gender difference in the anomalies.

Chapter 5

MATERIALS, METHODS, AND SUBJECTS

In this chapter, materials including equipment that was used in this research, subjects' preparation and selection criteria, sample size of the subjects and the method adopted for arteriographic analysis was discussed.

5.1 MATERIALS

5.1.1 Instrumentation

In this study all arteriograms were performed by the consultant radiologist of the Prince of Wales Hospital, Dr. Michael Chan, using the Philips V3000 Digital Subtraction Angiographic (DSA) system (Philips Medical Systems, 5680 DA BEST, The Netherlands). This system was equipped with the followings :

- High-contrast fluoroscopy
- Trace-subtraction fluoroscopy
- Last Image Hold
- Digital acquisition
- Anatomically programmed radiography
- Synchronized power injection of contrast medium with image acquisition
- Real-time digital subtracted image acquistion
- Polarity inversion

- Moving mask or new mask function
- Landmarking function
- Pixel shift function
- Digital optical recording

5.1.2 Contrast medium and its injection

Non-ionic contrast medium Iopamiro 150 was used in the study. All contrast injections were performed using the automatic injector, Angiomat 6000 of the Liebel-Flarsheim Company (2111 East Galbraith Road, Cincinnati, Ohio 45215 U.S.A.). The injection volume was 30ml per injection and the rate used was 8ml per second for the coeliac arteriogram and 6ml per second for the superior mesenteric arteriogram with optimization of contrast to reach region of interest. Injection pressure was automatically adjusted with the maximum set at 400psi. No patient suffered from any reactions due to the injection of the contrast medium.

5.2 METHODS

5.2.1 Patient preparation

All patients were prepared as follows:

1. The patients were nil by mouth for 4 hours before the procedure.

2. For each patient, an IV drip (on left side) was set up to keep the patient well hydrated.

3. Suitable measures were taken in diabetic patients to prevent the development of hypoglycaemia or hyperglycaemia.

4. Measures were taken to confirm that female patients of child bearing age were not pregnant on the day of the procedure.

5. Both groin areas of each patient were prepared (shaved and cleaned) for arterial puncture.

6. Evidence of coagulation defect was checked and corrected if necessary prior to the procedure.

7. The patients (and/or patients' relatives) were fully informed as to the nature and risk of the procedure and signed consents were obtained from patients (and/or patients' relatives).

8. In asthmatic patients and in patients with significant history of allergy, prophylactic steroid was given.

9. Patient emptied his/her bladder before coming to the angiographic suite.

5.2.2 Cathterization & Filming

Cathterization was performed with the Seldinger technique. The tip of the angiographic catheter was positioned in the origin of the corresponding vessel of

interest and the appropriate arteriograms were obtained. The filming sequence used in each series was '2 frames/second for 3 seconds, 1 frame/second for 2 seconds, then 1 frame at alternate second until the 23rd second'. No extra irradiation was incurred for hard copy documentation for this study.

5.2.3 Method of arteriographic analysis

5.2.3.1 Retrospective image retrieval

This study included a series of cases from January of 1995 to December of 1995. Arteriograms of subjects were retrieved from the system's hard disc or from archived optical disc files. Retrieved arteriographic images were all viewed on the viewing console of the V3000 System in digital subtracted positive format. The first image (i.e. the 0.5th second) was used as the mask and the second to fourth images were analysed. Standard magnification factor (x 4) were used.

5.2.3.2 Arterial variant analysis

After selecting the representative arteriograms, the arterial patterns were analysed with special attention to the arterial origins.

The vessels studied were the coeliac, common hepatic, proper hepatic, right hepatic, middle hepatic, left hepatic, gastroduodenal, right gastric, left gastric, and splenic artery. The possible origins of the above arteries are listed below.

1 Coeliac axis

(i) Aorta (Ao)

(ii) Superior Mesenteric Artery (SMA)

(iii) Absent (Ab)

2 Common hepatic artery

(i) Coeliac axis (Coeliac)

(ii) Superior Mesenteric Artery (SMA)

(iii) Aorta (Ao)

3 Proper hepatic artery

(i) Common hepatic artery (CH)

(ii) Absent (Ab)

4 Right hepatic artery

(i) Proper hepatic artery (PH)

(ii) Common hepatic artery (CH)

(iii) Superior Mesenteric Artery (SMA)

(iv) Accessory from SMA (ACC fr SMA)

(v) Accessory from gastroduodenal artery (ACC fr GD)

(vi) Absent (Ab)

5 Middle hepatic artery

(i) Right hepatic artery (RH)

(ii) Left hepatic artery (LH)

(iii) Right and Left hepatic (RH & LH)

(iv) Absent (Ab)

(v) Proper hepatic artery (PH)

(vi) Common hepatic artery (CH)

(vii) Gastroduodenal artery (GD)

6 Left hepatic artery

(i) Proper hepatic artery (PH)

(ii) Common hepatic artery (CH)

(iii) Left gastric artery (LG)

(iv) Accessory from left gastric artery (ACC fr LG)

(v) Coeliac axis (Coeliac)

(vi) Absent (Ab)

7 Gastroduodenal artery

(i) Common hepatic artery (CH)

(ii) Right hepatic artery (RH)

(iii) Left hepatic artery (LH)

(iv) Coeliac axis (Coeliac)

8 Right gastric artery

(i) Proper hepatic artery (PH)

(ii) Left hepatic artery (LH)

(iii) Right hepatic artery (RH)

(iv) Common hepatic artery (CH)

(v) Gastroduodenal artery (GD)

(vi) Absent (Ab)

(vii) Middle hepatic artery (MH)

(viii) Bifurcation of proper hepatic artery (BIF of PH)

(ix) Bifurcation of common hepatic artery (BIF of CH)

9 Left gastric artery

(i) Coeliac axis (Coeliac)

(ii) Aorta (Ao)

(iii) Bifurcation of coeliac axis (BIF of Coeliac)

(iv) Absent (Ab)

10 Splenic artery

(i) Aorta (Ao)

(ii) Superior Mesenteric Artery (SMA)

5.2.3.3 Data sheet

In order to facilitate data recording for future analysis, the possible variants of the ten arteries studied are charted up in the sample data sheet shown in figure 5.01.

The vertical row represents the ten arteries studied and their respective possible origins and the horizontal row denotes the individual subjects (in assigned numbers). Any identification of one possible origin of the vessels studied would be entered as 'one' and after counting all 276 subjects, the sum of each category of possible origin would be calculated. From the numbers, further calculation of the percentages of each listed possible origin of the vessels studied would be made.

5.2.3.4 Radiographic images and trace drawings

All illustrative arteriograms shown in this thesis were scanned from the original digital subtracted arteriograms using the HP 4P Desk Scanner. All trace diagrams were manually traced and coloured by the author.

Figure 5.01 Sample Data Sheet

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
Ref. No.																															
SEX - M																															
AGE																															
HCC																															
COELIAC	Ao																														
	SMA																														
	Ab																														
COMMON HEPATIC	Coeliac																														
	SMA																														
	Ao																														
PROPER HEPATIC	CH																														
	Ab																														
RIGHT HEPATIC	PH																														
	CH																														
	SMA																														
	ACC fr SMA																														
	ACC fr GD																														
	Coeliac																														
	ACC fr PH																														
MIDDLE HEPATIC	RH																														
	LH																														
	RH & LH																														
	Ab																														
	PH																														
	CH																														
	GD																														
LEFT HEPATIC	PH																														
	CH																														
	LG																														
	ACC fr LG																														
	Coeliac																														
	Ab																														
GASTRO - DUO-DENAL	CH																														
	RH																														
	LH																														
	Coeliac																														
RIGHT GASTRIC	PH																														
	LH																														
	RH																														
	CH																														
	GD																														
	Ab																														
	MH																														
	BIF of PH																														
	BIF of CH																														
LEFT GASTRIC	Coeliac																														
	Ao																														
	BIF of Coeliac																														
	Ab																														
SPLENIC	Coeliac																														
	Ao																														
	SMA																														

5.3 SUBJECTS

5.3.1 Basic inclusion criteria

All subjects included were clinically suspected to have abdominal masses or gastro-intestinal problems. Patients were collected at random with no sex predilection. Subjects who had abdominal operations were excluded since any abdominal operation might alter the abdominal vascular pattern.

5.3.2 Confirmation criteria

The subjects included were either suffering from HCC, gastro-intestinal bleeding or other abdominal tumours. Confirming diagnosis were based on the following criteria.

5.3.2.1 Hepatocellular carcinoma (HCC) (n= 166)

- Positive hepatitis B surface antigen (+ HBsAg) (n=166)
- Raised alpha feto-protein level (equal or greater than 500 ng/ml) (n=166)
- Presence of solitary or query multiple focal hepatic lesion on USG (n=166)
- Presence of solitary or query multiple focal hepatic lesion on CT scan with or without lipiodol (n=166)
- Presence of solitary or query multiple focal hepatic lesion on arteriography with or without lipiodol (n=166)
- Histo-pathological tissue confirmation (n=41)

5.3.2.2 Gastrointestinal problems (n=105)

- Clinical suspicion of internal bleeding particularly the gastrointestinal origin (n=105)
- Positive 99m-Technitium labelled Red Blood Cell scan (n=5)
- Normal alpha feto-protein level (equal or smaller than 10 ng/ml (n=100)
- Absence of solitary or multiple focal hepatic lesion on USG (n=105)
- Absence of solitary or multiple focal hepatic lesion on arteriography without lipiodol (n=105)
- Presence of leakage of contrast on arteriography (n=4)

5.3.2.3 The remaining five subjects :

Two subjects had metastases with multiple hepatic lesions on USG scan and known primary malignancies.

Two subjects had cavernous haemangioma with characteristic arteriographic appearance and normal alpha feto-protein level (equal or smaller than 10 ng/ml).

One subject had a retro-peritoneal mass.

5.3.3 Final number of subjects recruited

From January of 1995 to Feburary of 1996, 276 subjects were recruited, and 197 were males and 79 were females. Age ranged from 17 to 92 with a median of 54±13 years. 166 subjects were having HCC and 110 subjects with no HCC.

Chapter 6

RESULTS

From January of 1995 to Feburary of 1996, the upper abdominal angiograms of 276 subjects were analysed. Of the 276 subjects (figure 6.01 & figure 6.02)), 197 were males and 79 were females; and 166 subjects were diagnosed to have HCC with the other 110 subjects having gastro-intestinal problems or other upper abdominal lesions. Age ranged from 17 to 92 with a median of 54±13 years. When tested with the Kolmogorov-Smirnov Goodness of Fit Test, the age distribution frequency was a normal distribution (figure 6.03) with a 2-tailed p value of 0.06 which is greater than 0.05.

In this chapter, the percentages of the variants observed for each vessel studied are presented. Corresponding illustrations are displayed with the variants coloured.

Figure 6.01 Proportion of males to females

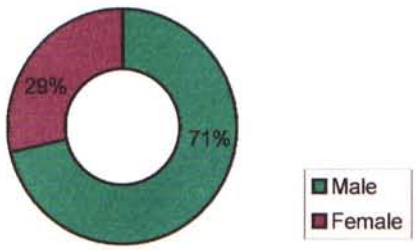


Figure 6.02 Proportion of subjects with HCC to those without HCC

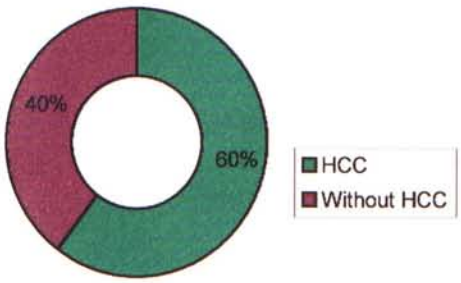


Figure 6.03 Frequency distribution of age

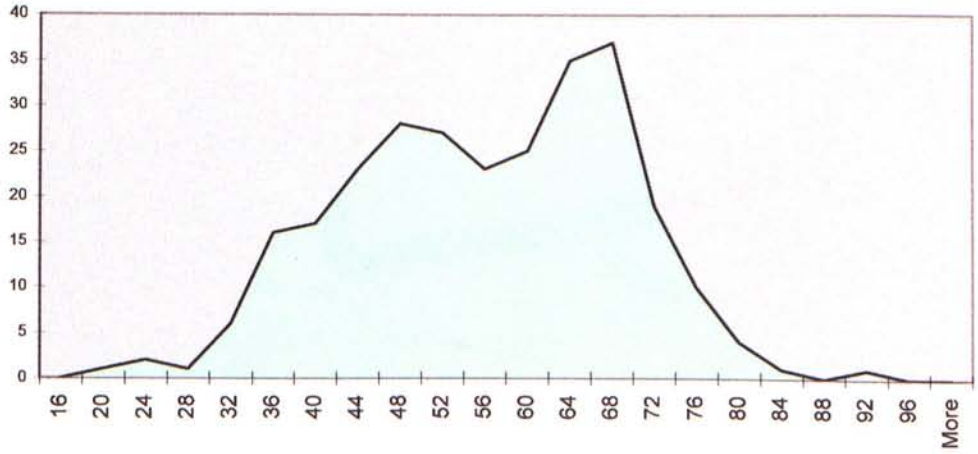


Figure 6.04a Schematic drawing showing classical presentation of the coeliac axis

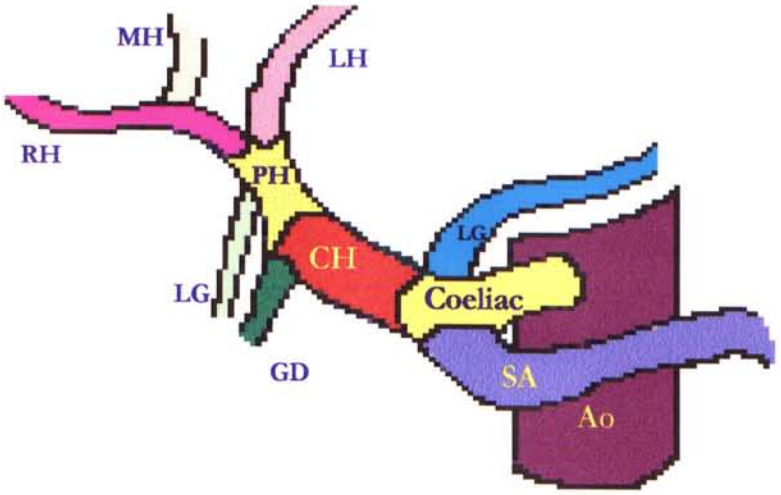


Figure 6.04b Radiograph showing classical presentation of the coeliac axis

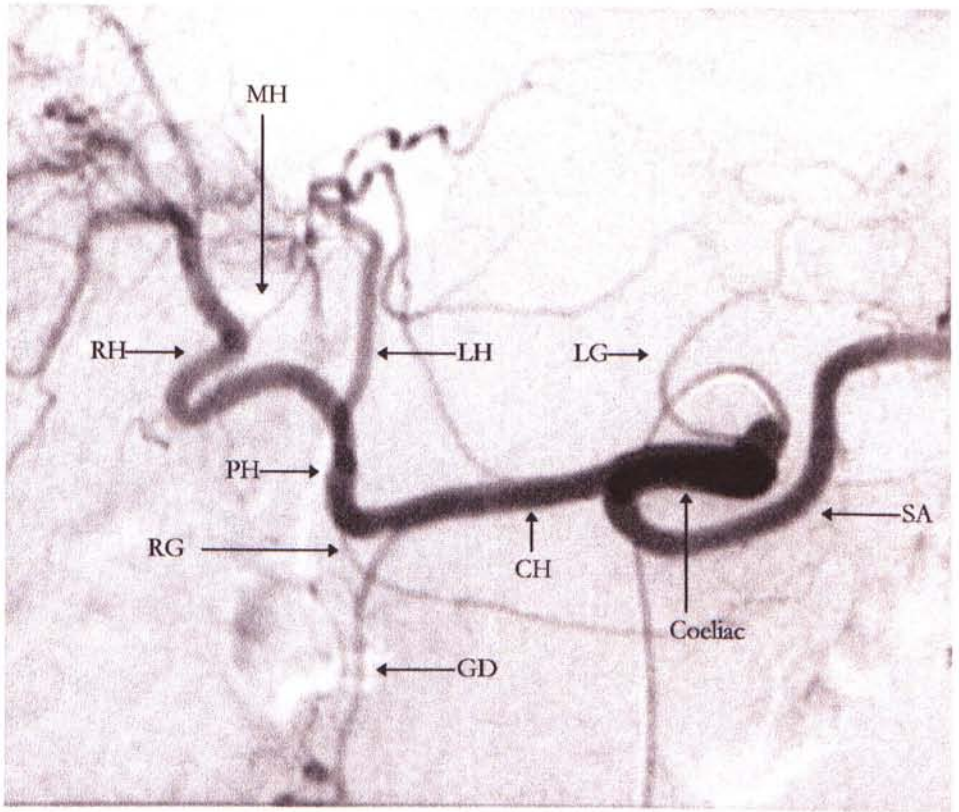


Figure 6.05a Radiograph showing classical trifurcation of the coeliac axis

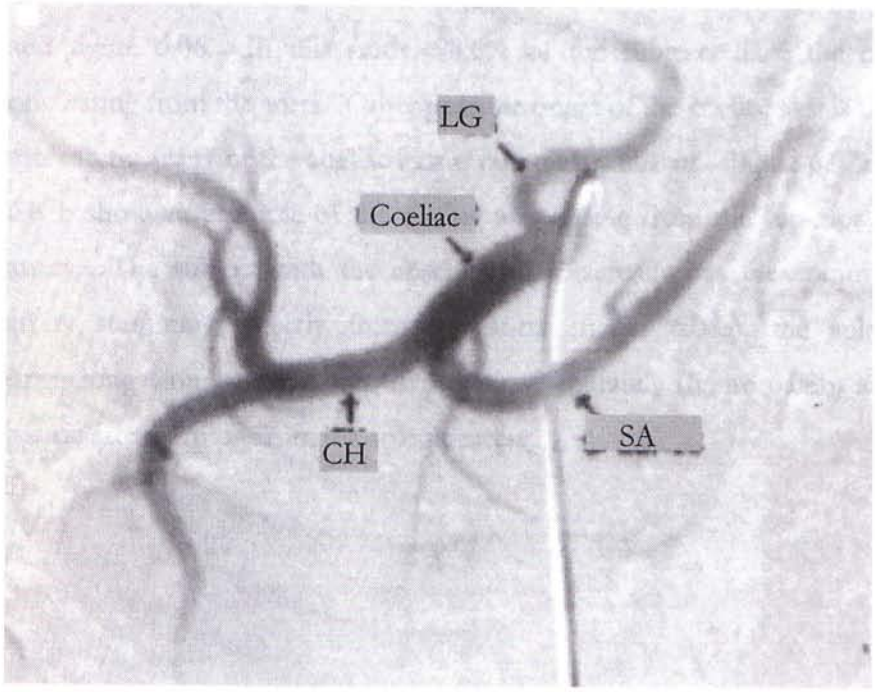
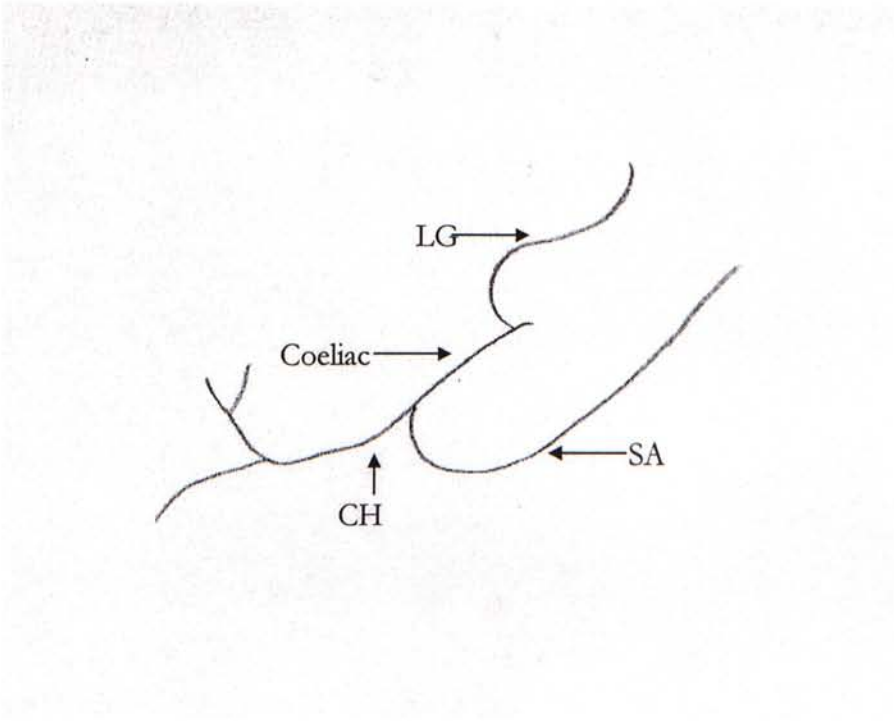


Figure 6.05b Schematic drawing showing classical trifurcation of the coeliac axis



6.1 Coeliac axis

The classical presentation of the coeliac axis is from the aorta (figure 6.04a). The observed percentages of the origin of the coeliac axis is listed in table 6.01 and figure 6.06. In this study, 98.6% of the subjects have the coeliac axis originating from the aorta. Other possible origin of the coeliac axis is the superior mesenteric artery or the coeliac axis is completely absent. Figure 6.07a and figure 6.07b show an example of the coeliac axis arising from the superior mesenteric artery. The subject with the absent coeliac actually has the common hepatic artery stemming directly from the aorta (figure 6.08a), the splenic artery originating from another site of the aorta separately (figure 6.08b) and the left gastric artery arising from the splenic artery.

Table 6.01 Origin of the coeliac axis

Vessel origin	Number observed	Percentage	Illustration
Aorta	272	98.6%	figure 6.04
Superior mesenteric artery	3	1.1%	figure 6.07a, 6.07b
Absent	1	0.4%	figure 6.08a, 6.08b

Figure 6.06 Origin of the coeliac axis

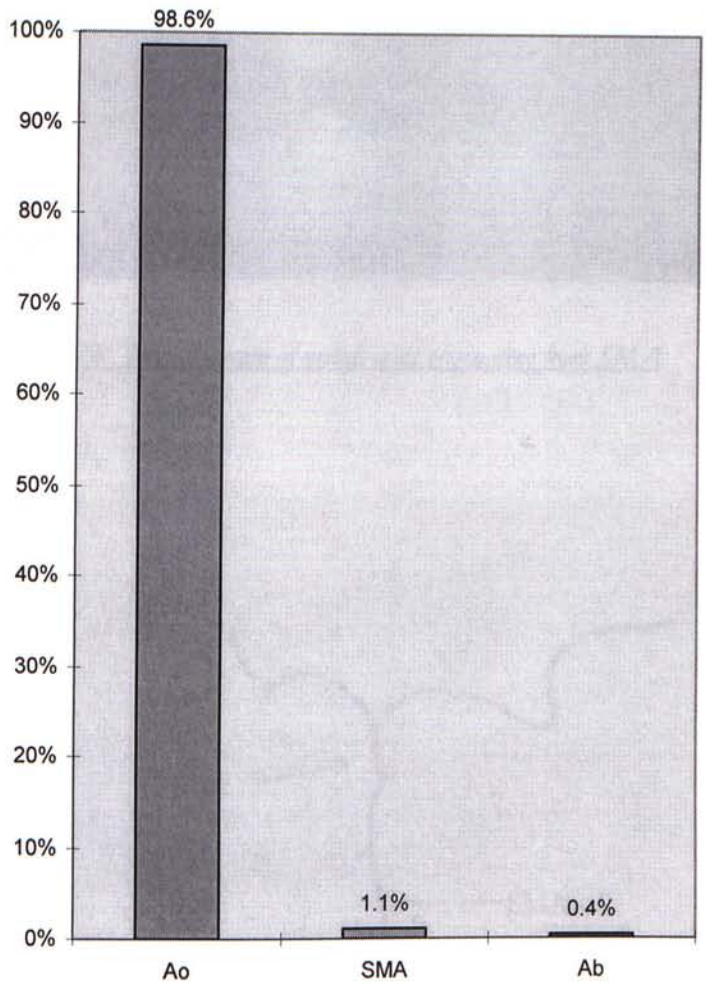


Figure 6.07a Coeliac axis originate from SMA

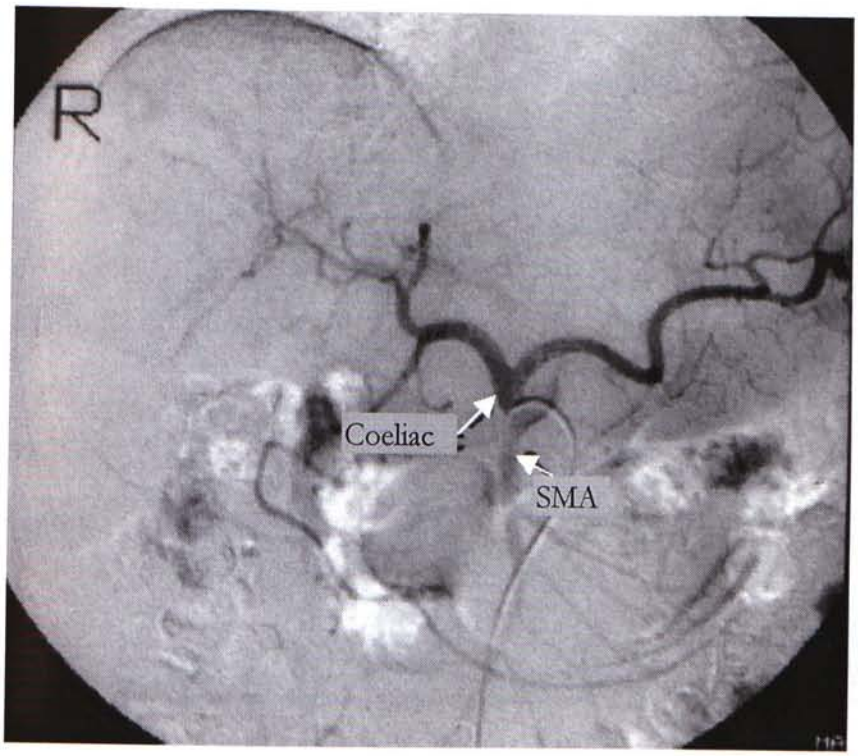


Figure 6.07b Trace diagram of coeliac axis originating from SMA

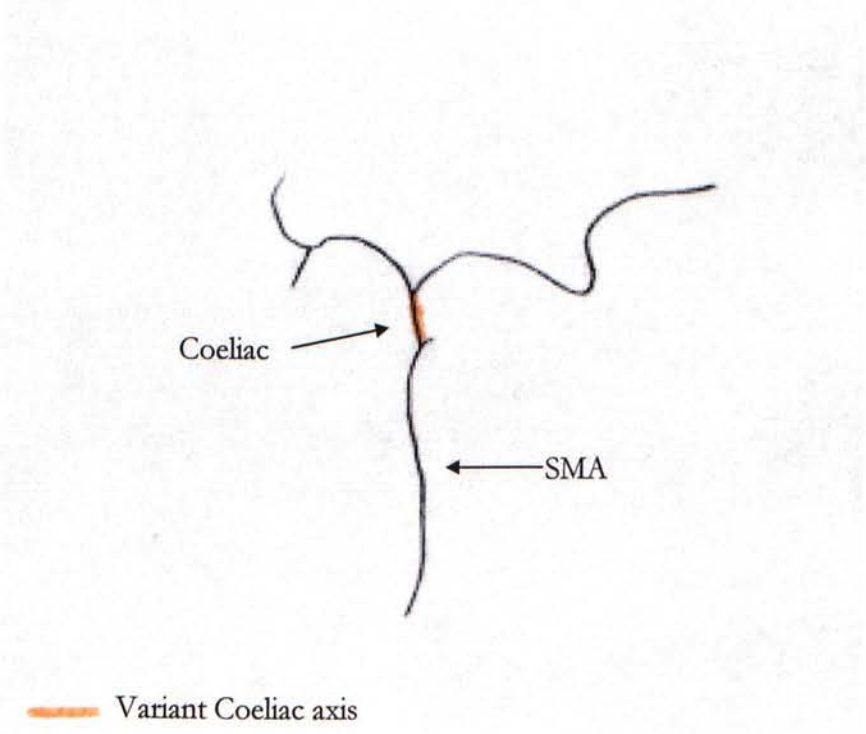


Figure 6.08a CH fr Ao

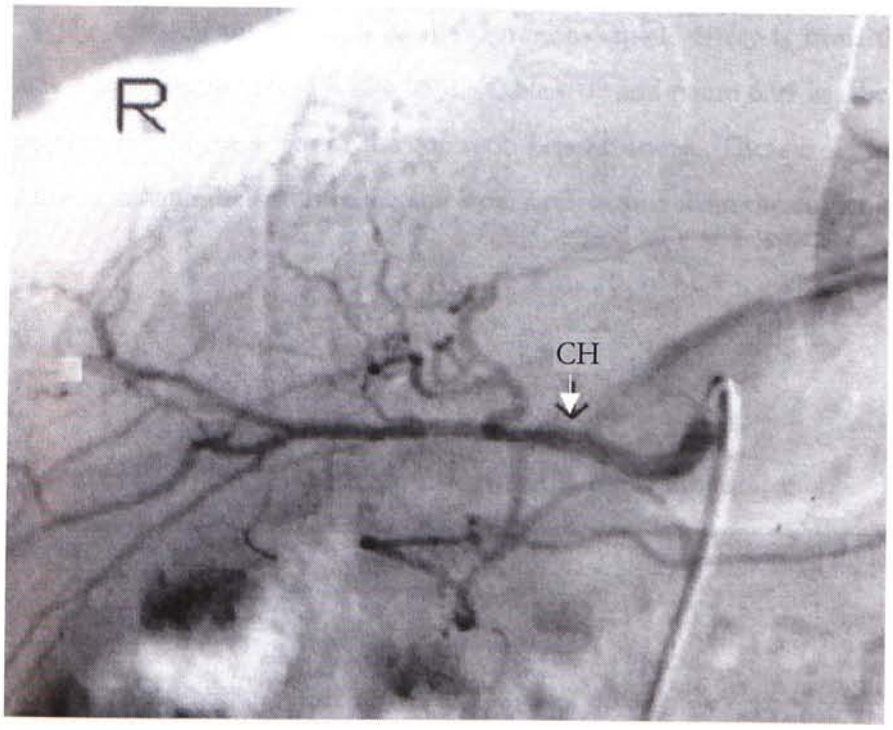
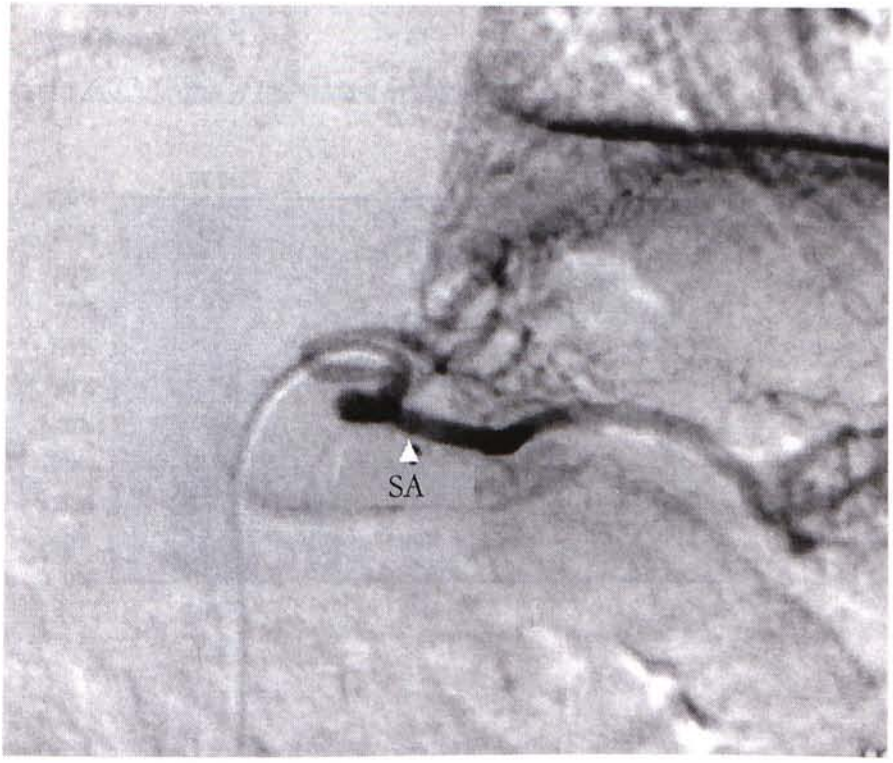


Figure 6.08b SA fr Ao



6.2 Common hepatic artery

The classical presentation of the common hepatic artery is from the coeliac axis (figures 6.04a & 6.05a & 6.05b). Table 6.02 and figure 6.09 list the observed percentages of the origin of the common hepatic artery. There is a 97.8% of the subjects having the common hepatic artery originating from the coeliac axis.

Table 6.02 Origin of the common hepatic artery

Vessel origin	Number observed	Percentage	Illustration
Coeliac axis	270	97.8%	figure 6.05a, 6.05b
Superior mesenteric artery	4	1.4%	figure 6.10a, 6.10b
Aorta	2	0.7%	figure 6.08a

Figure 6.09 Origin of the common hepatic artery

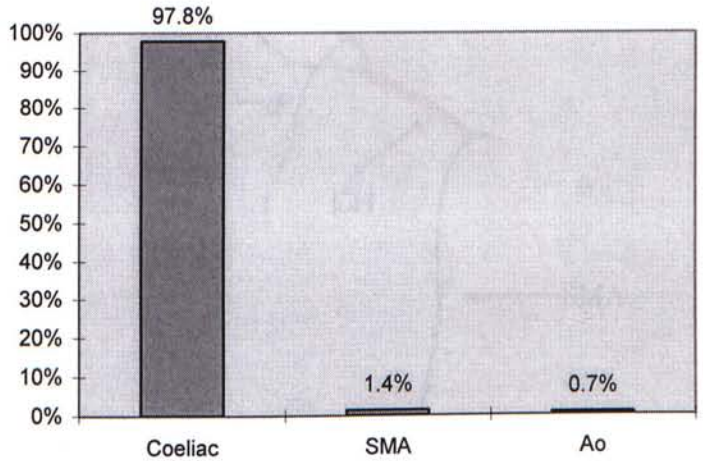


Figure 6.10a CH fr SMA (Absent PH, RH fr CH, MH fr LH)

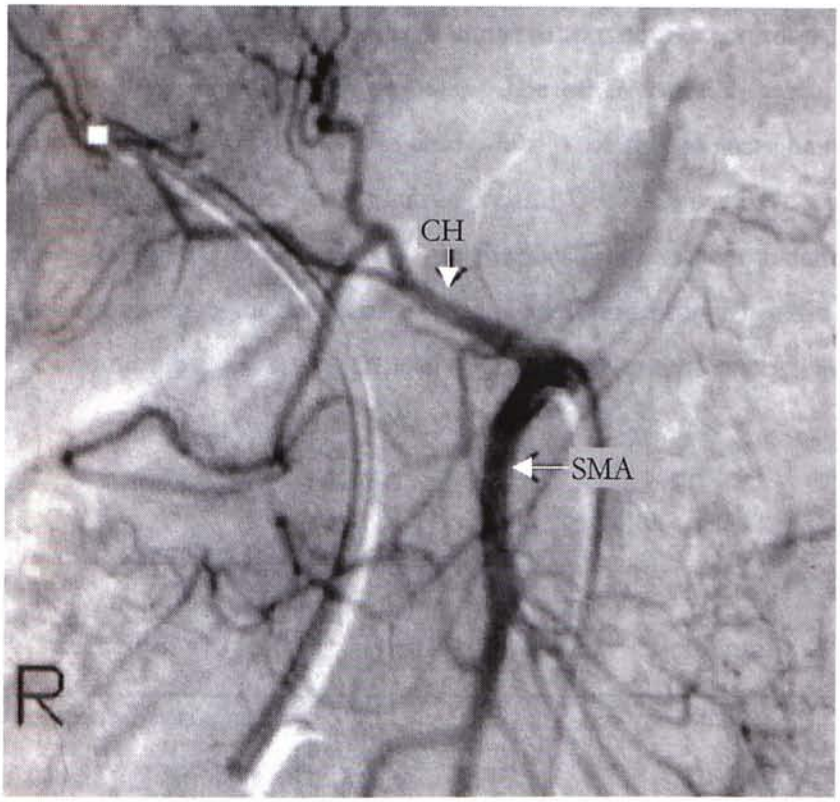
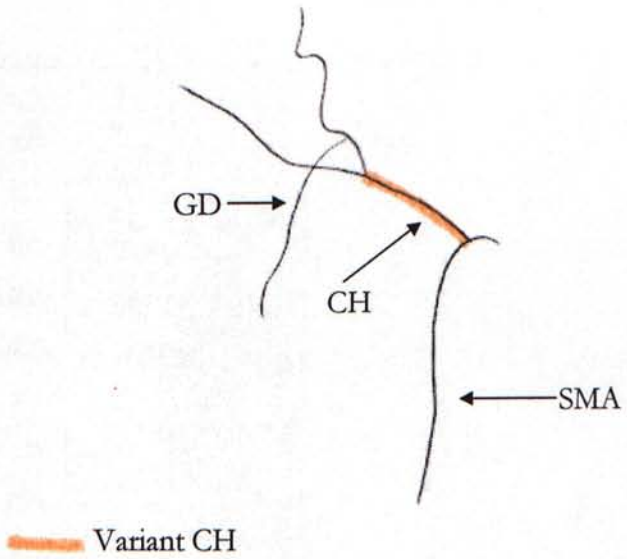


Figure 6.10b Schematic drawing of a 'CH fr SMA (Absent PH)'



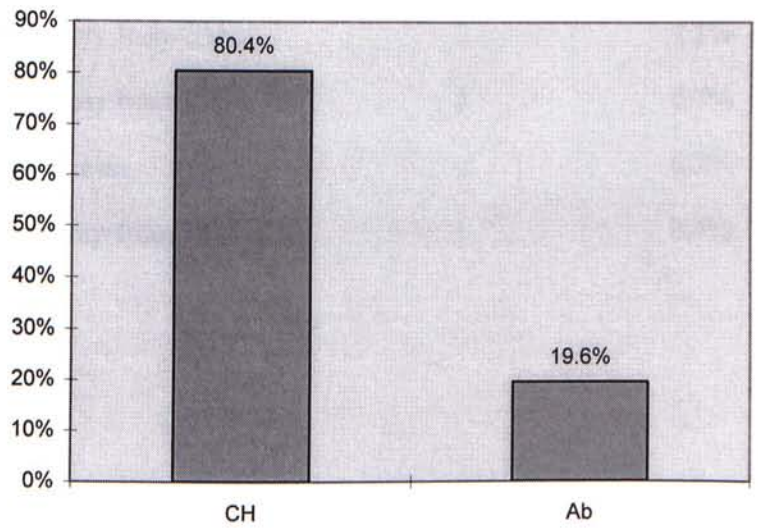
6.3 Proper hepatic artery

The proper hepatic artery is a continuation of the common hepatic artery classically (figure 6.04 & figure 6.05). The other common variant is an absent proper hepatic artery. In the study, 80.4% of the subjects have the classical presentation of the proper hepatic artery and 19.6% do not have a proper hepatic artery (table 6.03 & figure 6.11). In the illustration shown in figure 6.10a, the subject presents with two variants, one is an absent proper hepatic and the other is a common hepatic arising from the superior mesenteric artery.

Table 6.03 Origin of the proper hepatic artery

Vessel origin	Number observed	Percentage	Illustration
Common hepatic artery	222	80.4%	figure 6.04, 6.05
Absent	54	19.6%	figure 6.10a, 6.10b

Figure 6.11 Origin of the proper hepatic artery



6.4 Right hepatic artery

The text book presentation of the right hepatic artery is from the proper hepatic artery (figure 6.04 & figure 6.05). In this study, 80.4% of the subjects have this text book presentation. The other observed variants of the origin of the right hepatic artery are from the common hepatic artery or the superior mesenteric artery. An accessory right hepatic can also arise from the superior mesenteric artery (figure 6.14a1 & 6.14a2 are arteriograms of the same subject demonstrating the presence of an accessory right hepatic originating from the superior mesenteric artery in addition to a right hepatic artery originating from the proper hepatic artery), or from the gastroduodenal artery, the coeliac axis, or the proper hepatic artery. The observed frequency of these variants are listed in table 6.04 and figure 6.12.

Table 6.04 Origin of the right hepatic artery

Vessel origin	Number observed	Percentage	Illustration
Proper hepatic artery	222	80.4%	figure 6.04, 6.05
Common hepatic artery	39	14.1%	figure 6.10a, 6.10b
Superior mesenteric artery	13	4.7%	figure 6.13a, 6.13b
Accessory from SMA	3	1.1%	figure 6.14a, 6.14b
Accessory from GD	2	0.7%	figure 6.15a, 6.15b
Coeliac axis	2	0.7%	figure 6.16a, 6.16b
Accessory from PH	1	0.4%	figure 6.17a, 6.17b

Figure 6.12 Origin of the right hepatic artery

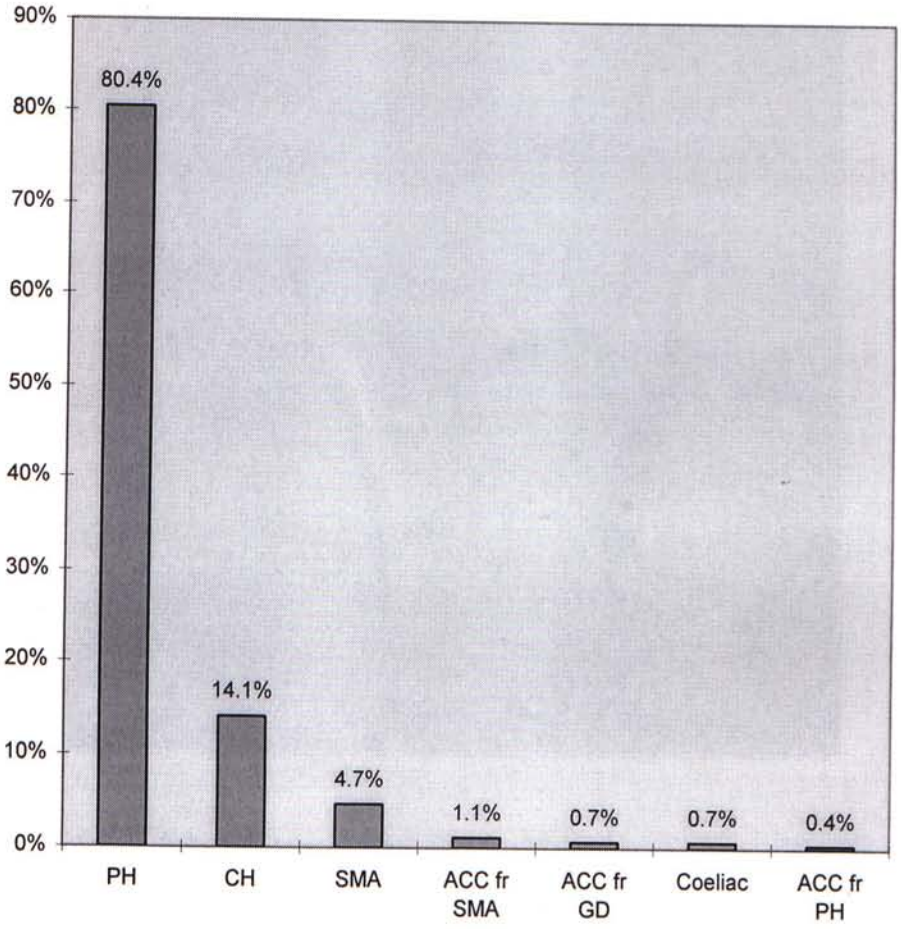


Figure 6.13a RH fr SMA

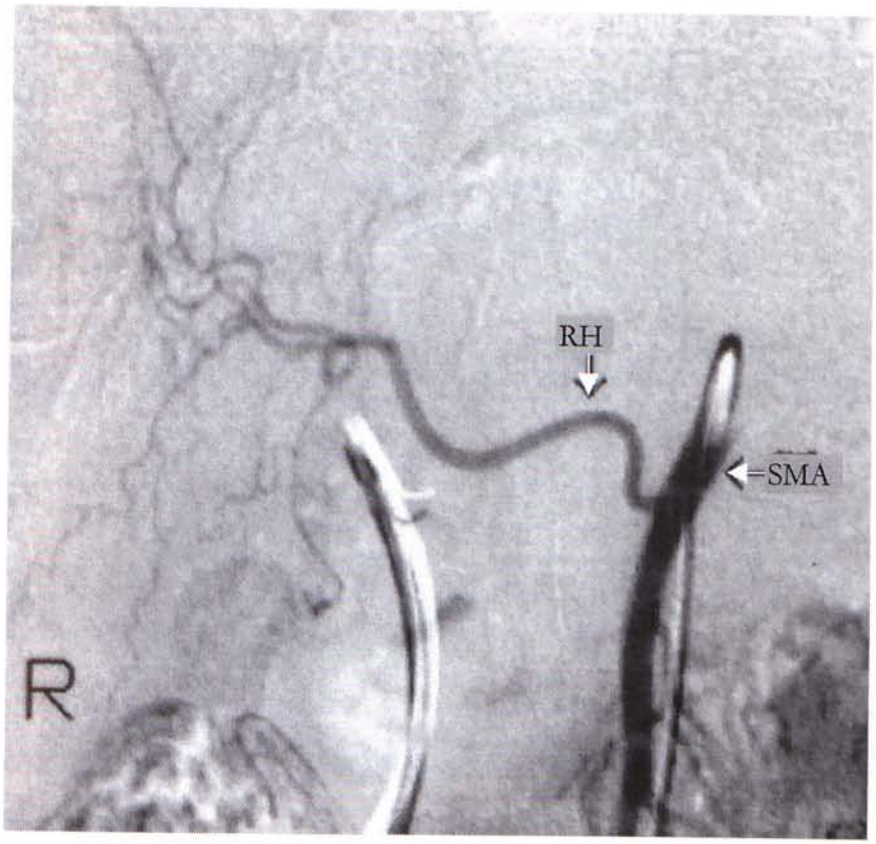


Figure 6.13b Schematic diagram showing 'RH fr SMA'

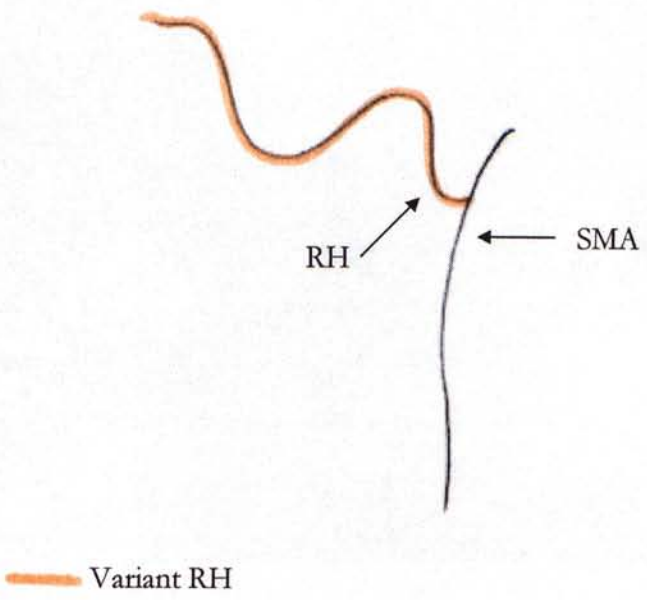


Figure 6.14a ACC RH fr SMA

Figure 6.14a1

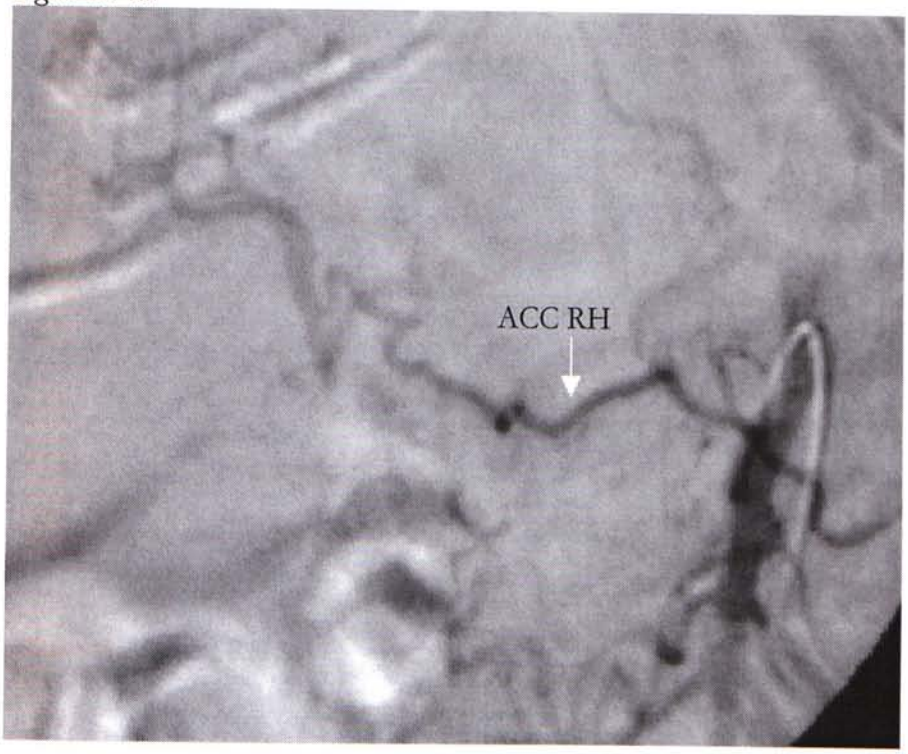


Figure 6.14a2

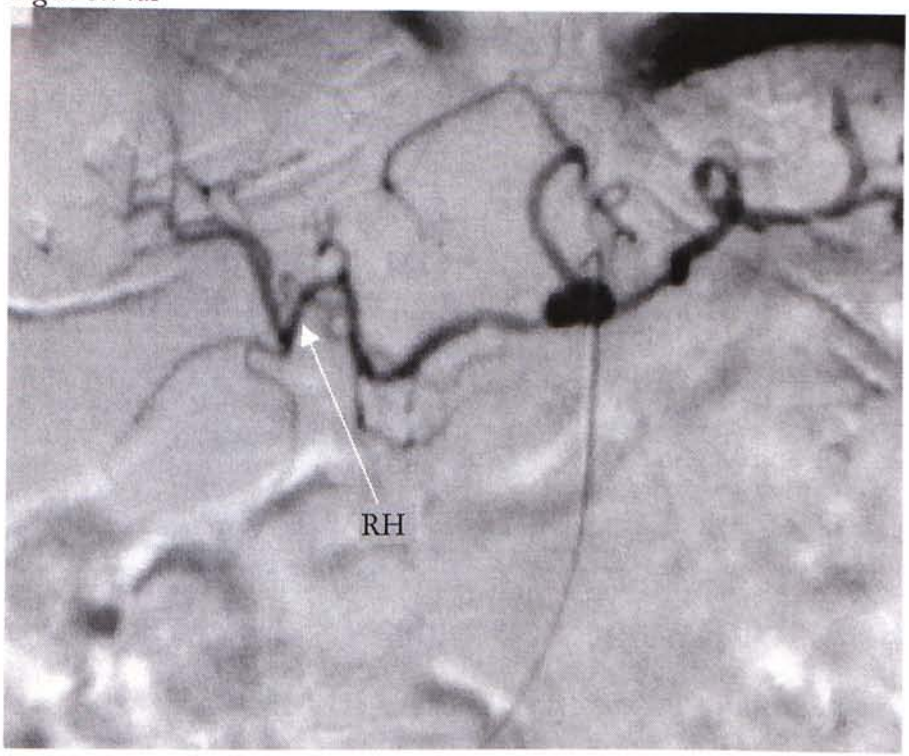


Figure 6.14b Schematic diagram showing 'ACC RH fr SMA'

Figure 6.14b1

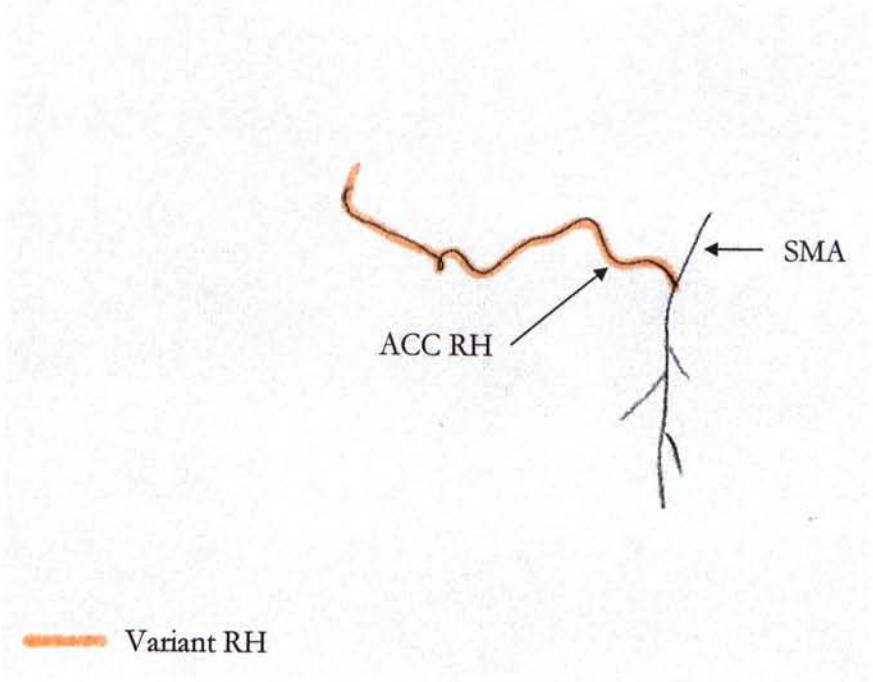


Figure 6.14b2

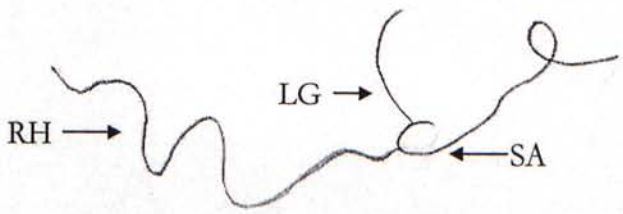


Figure 6.15a ACC RH fr GD

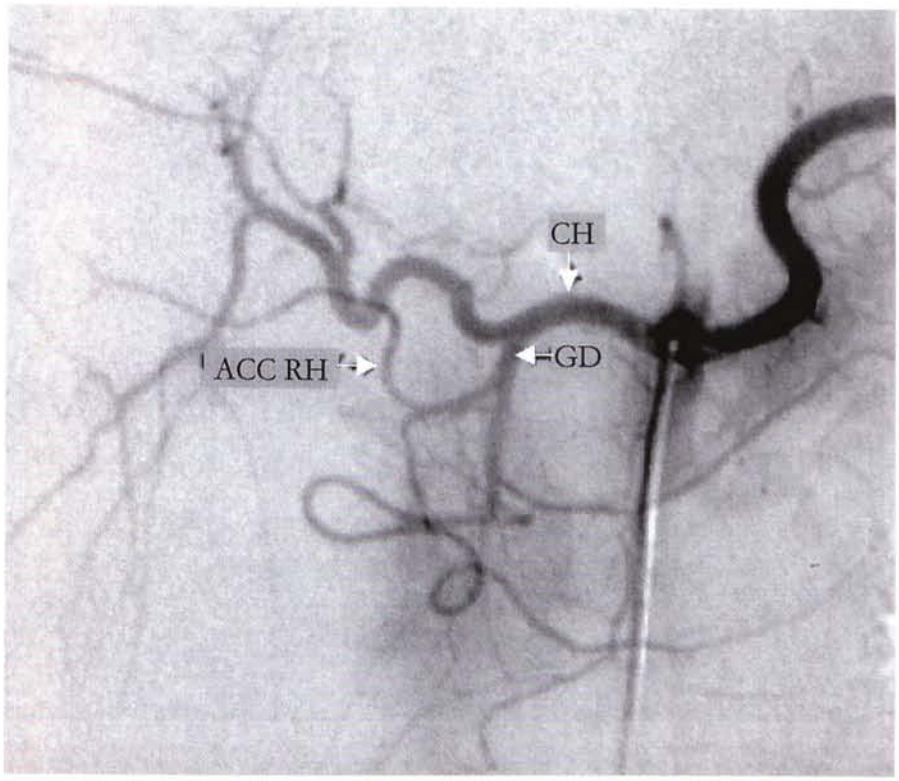


Figure 6.15b Schematic diagram showing 'ACC RH fr GD'

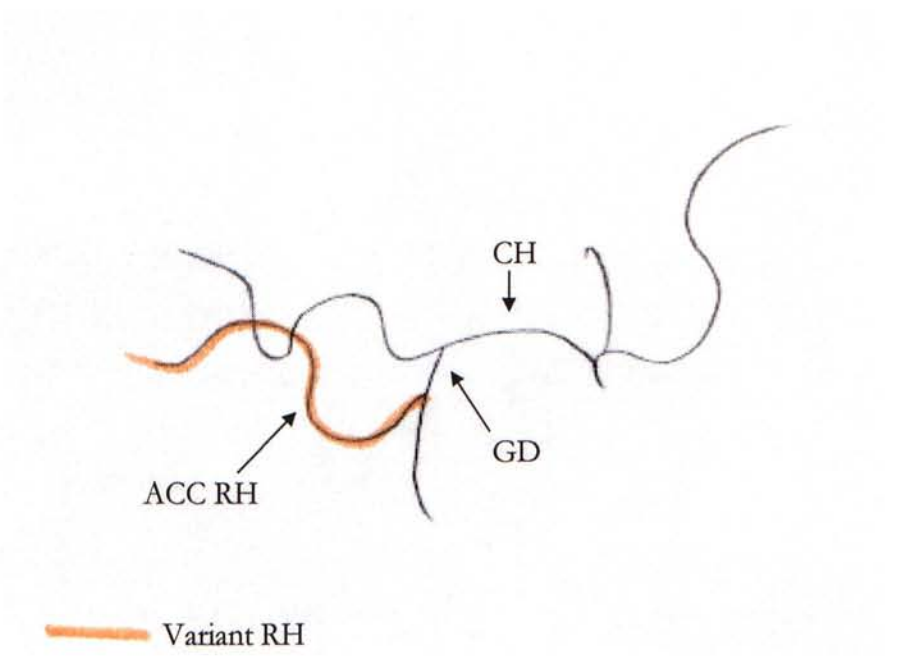


Figure 6.16a RH fr Coeliac axis

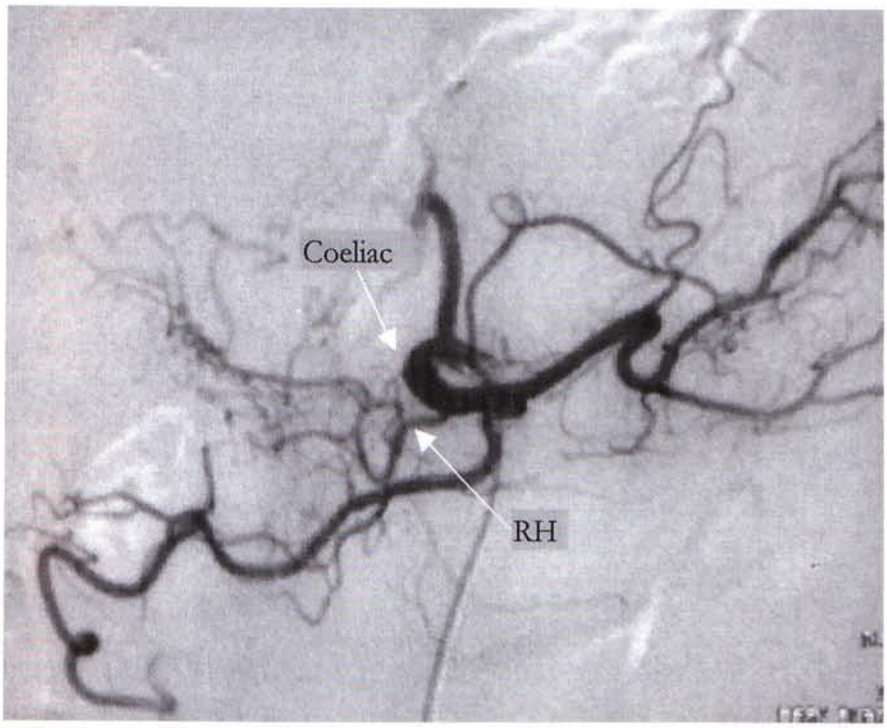


Figure 6.16b Schematic diagram showing 'RH fr Coeliac axis'

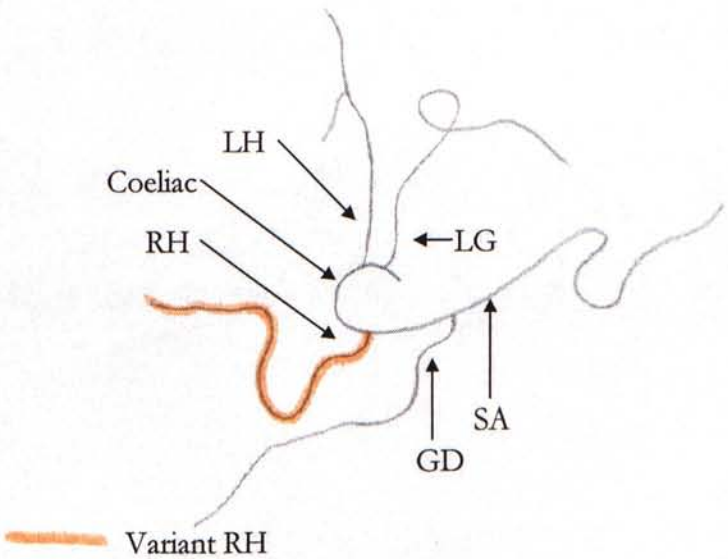


Figure 6.17a ACC RH fr PH

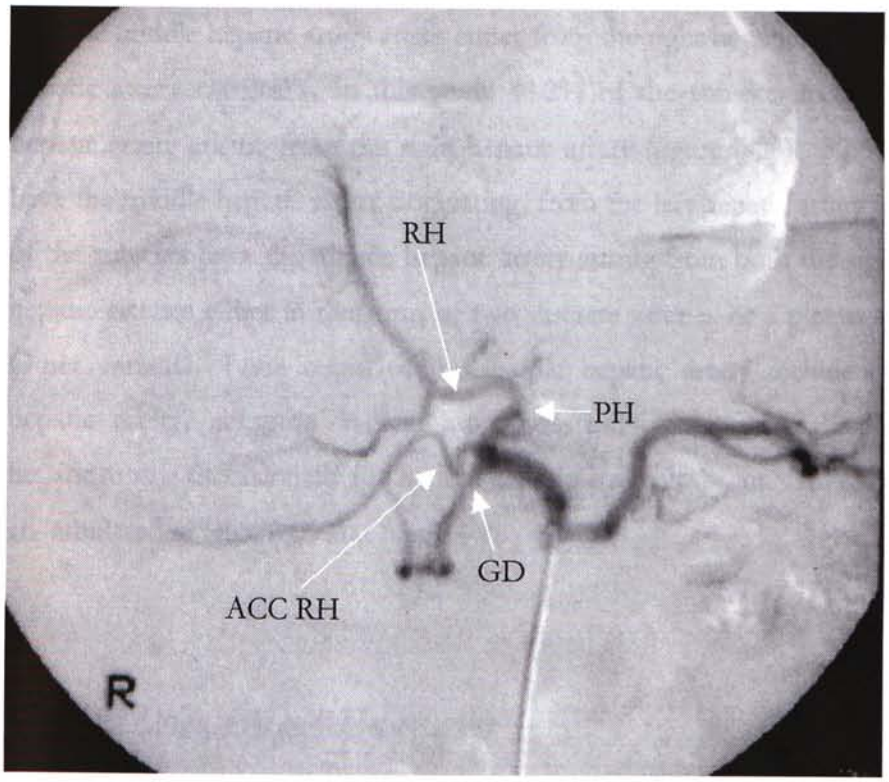
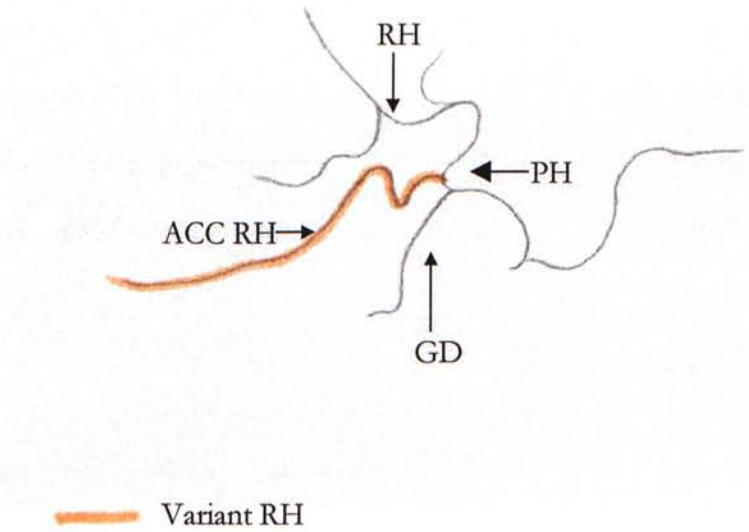


Figure 6.17b Schematic diagram showing 'ACC RH fr PH'



6.5 Middle hepatic artery

The middle hepatic artery arises either from the right hepatic artery or the left hepatic artery classically. In this study 44.2% of the subjects have the middle hepatic artery arising from the right hepatic artery (figure 6.04). 32.6% subjects have the middle hepatic artery originating from the left hepatic artery and 14.9% of the subjects have the middle hepatic artery arising from both the right and left hepatic arteries either in the form of two discrete arteries or a plexus of arteries. Other variants of the origin of the middle hepatic artery include the proper hepatic artery, common hepatic artery, gastroduodenal artery, accessory left hepatic artery. Occasionally the middle hepatic artery is absent. These occurrence are tabulated in table 6.05 and figure 6.18

Table 6.05 Origin of the middle hepatic artery

Vessel origin	Number observed	Percentage	Illustration
Right hepatic artery	123	44.6%	figure 6.19a, 6.19b
Left hepatic artery	90	32.6%	figure 6.20a, 6.20b
RH & LH	41	14.9%	figure 6.21a, 6.21b
Absent	14	5.1%	
Proper hepatic artery	5	1.8%	figure 6.22a, 6.22b
Common hepatic artery	2	0.7%	figure 6.23
Gastroduodenal artery	1	0.4%	figure 6.24

Figure 6.18 Origin of the middle hepatic artery

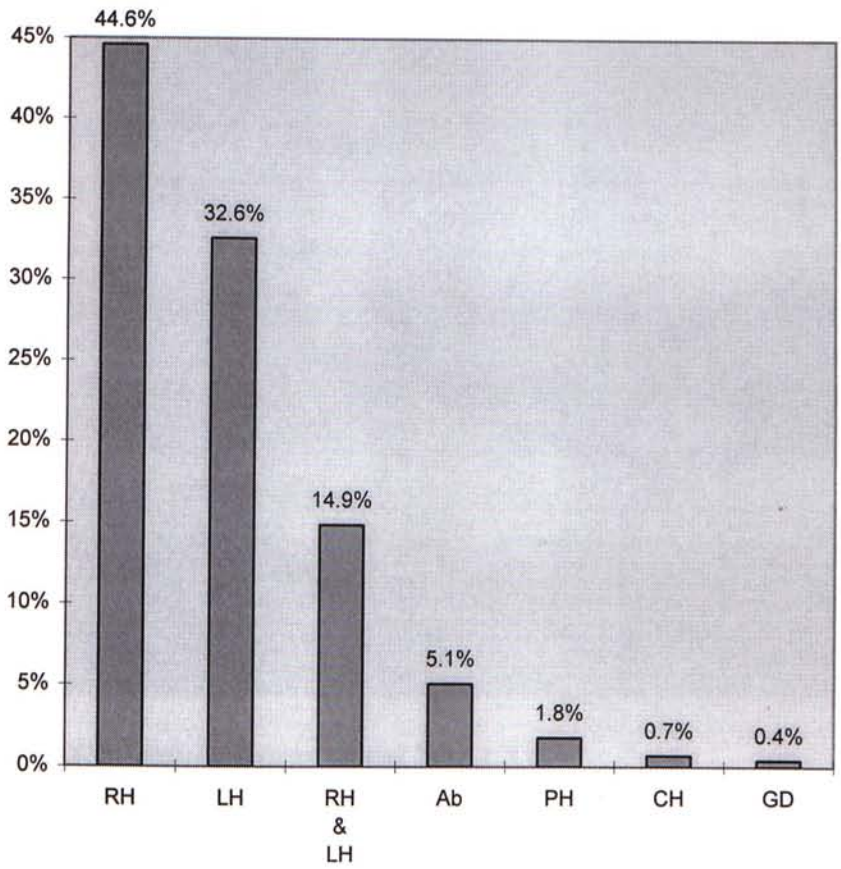


Figure 6.19a MH fr RH

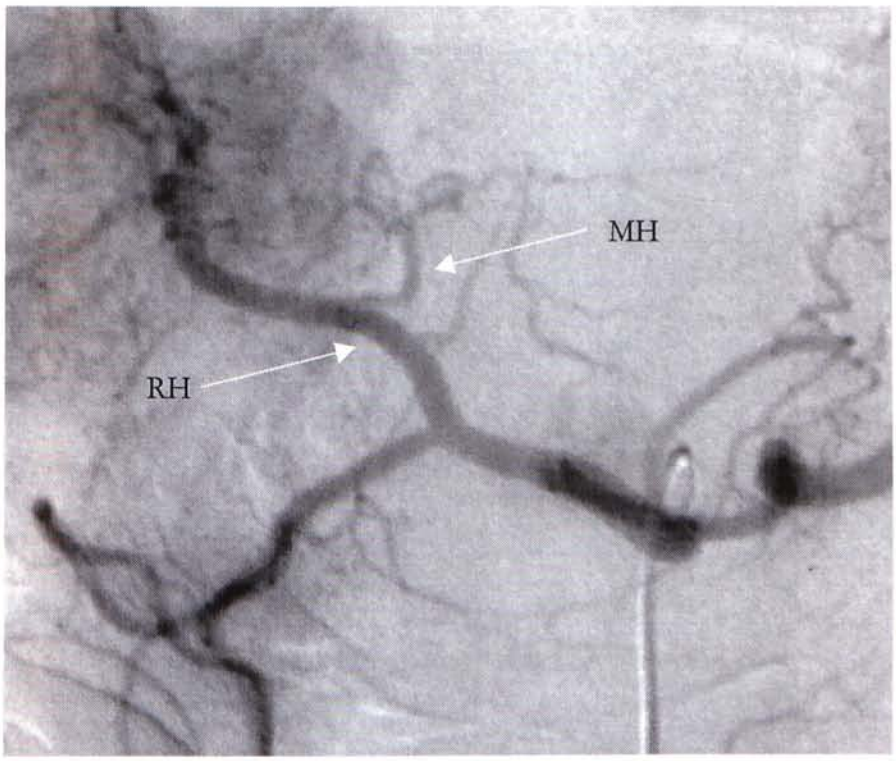


Figure 6.19b Schematic diagram showing 'MH fr RH'

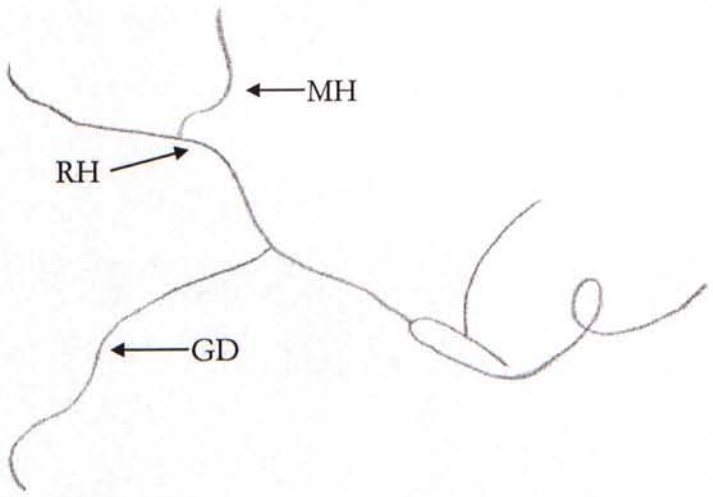


Figure 6.20a MH fr LH

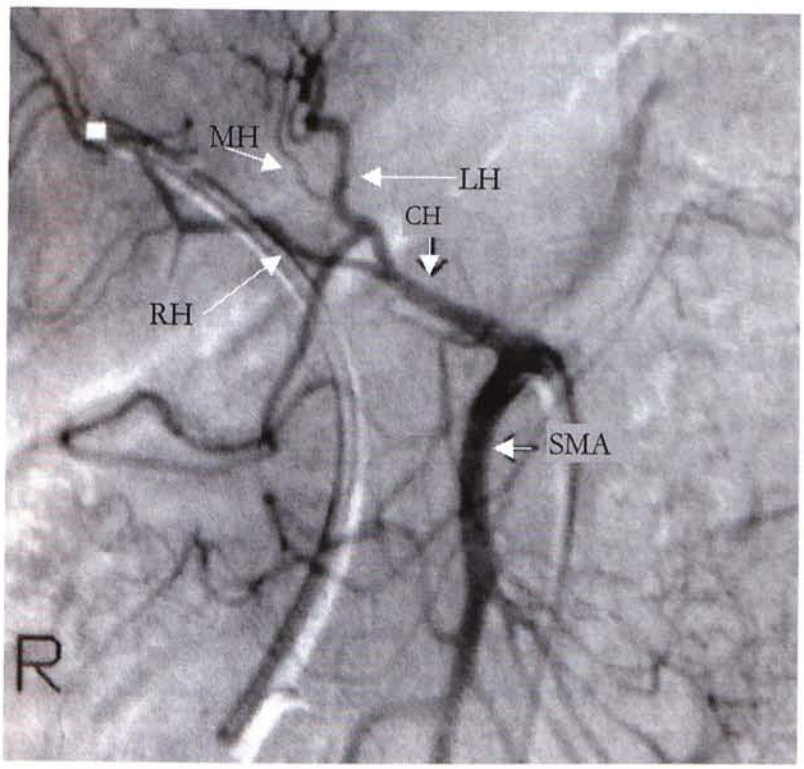


Figure 6.20b Schematic diagram showing 'MH fr LH'

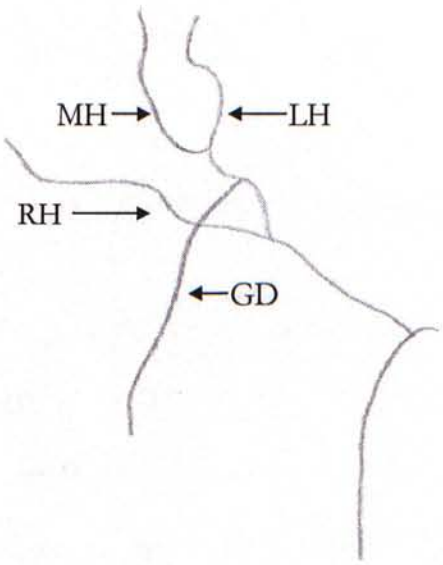


Figure 6.21a MH fr RH & LH

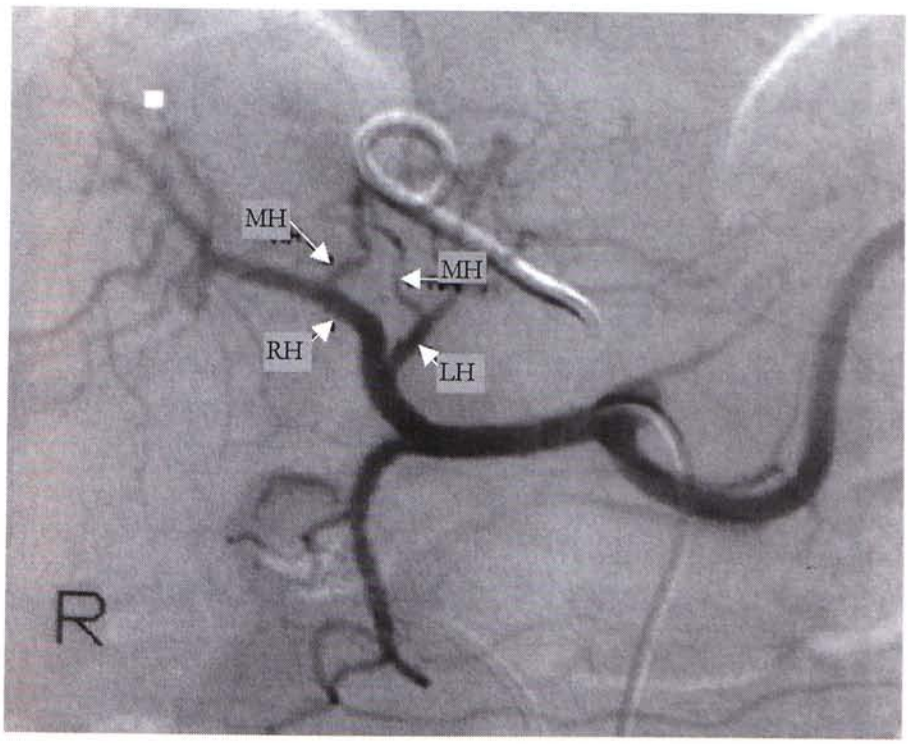
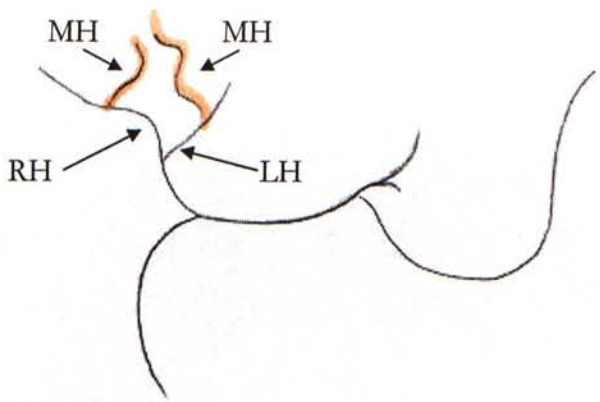


Figure 6.21b MH fr RH & LH



Variant MH

Figure 6.22a MH fr PH

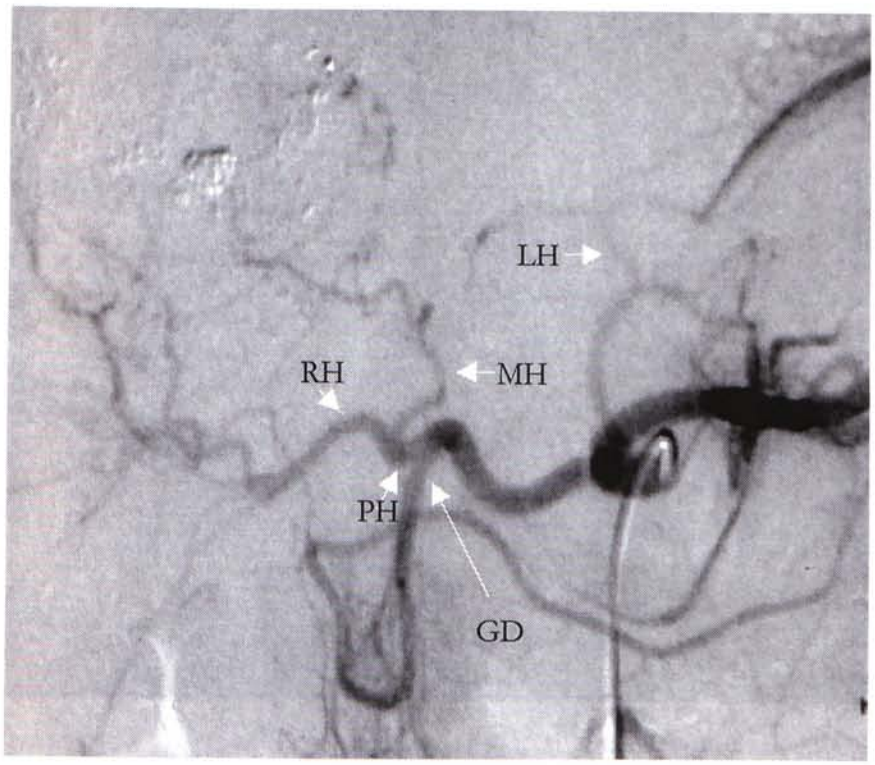


Figure 6.22b Schematic diagram showing 'MH fr PH'

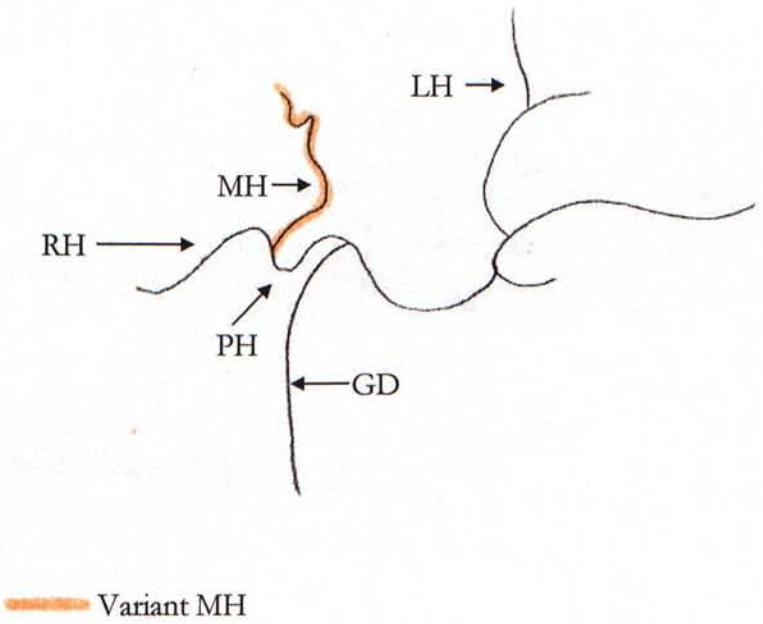


Figure 6.23a MH fr CH (RG fr MH)

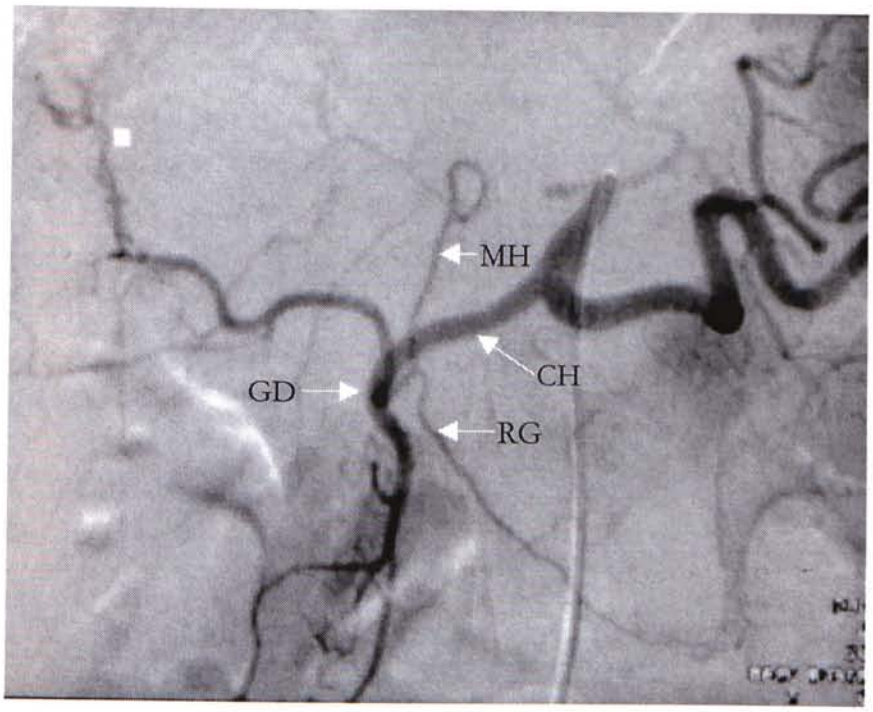


Figure 6.23b Schematic diagram showing 'MH fr CH (RG fr MH)'

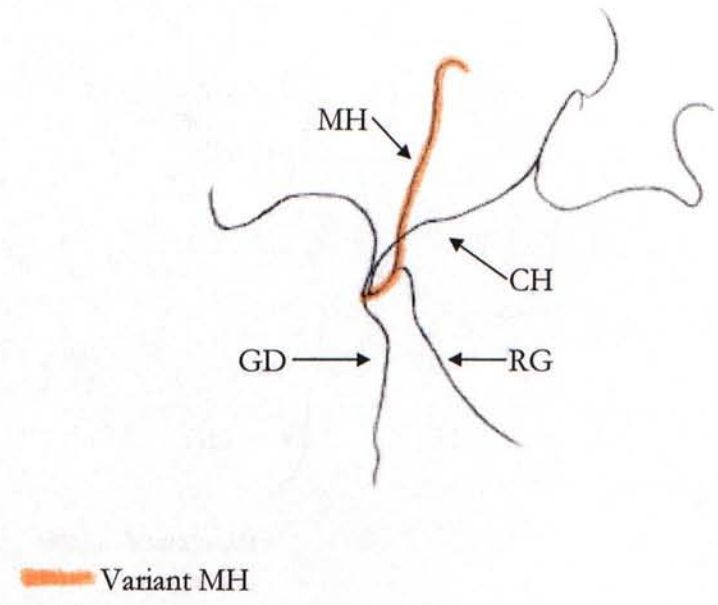


Figure 6.24a MH fr GD

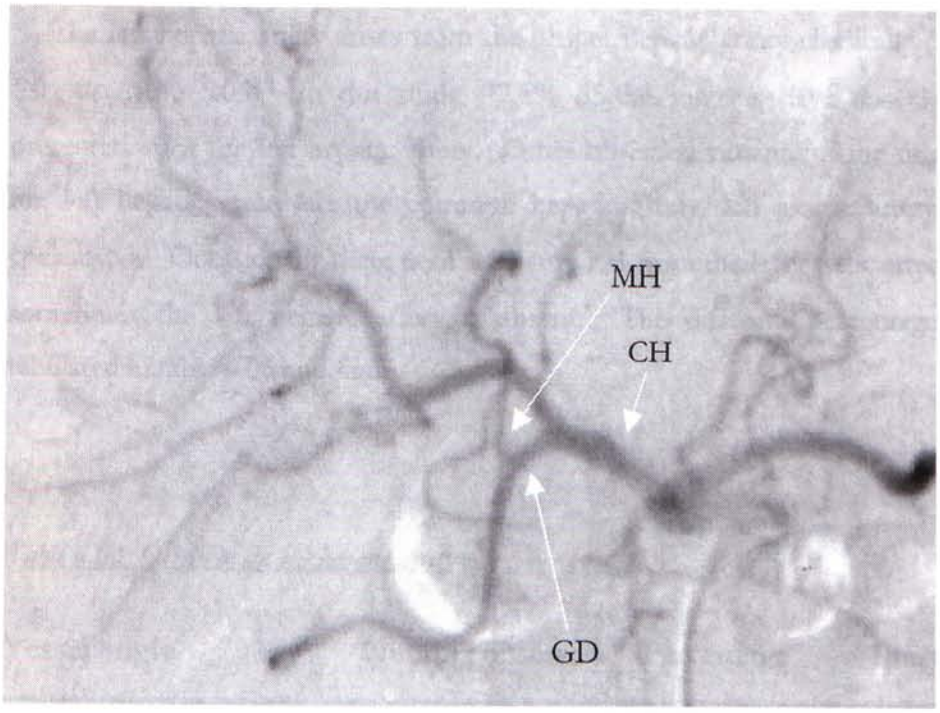
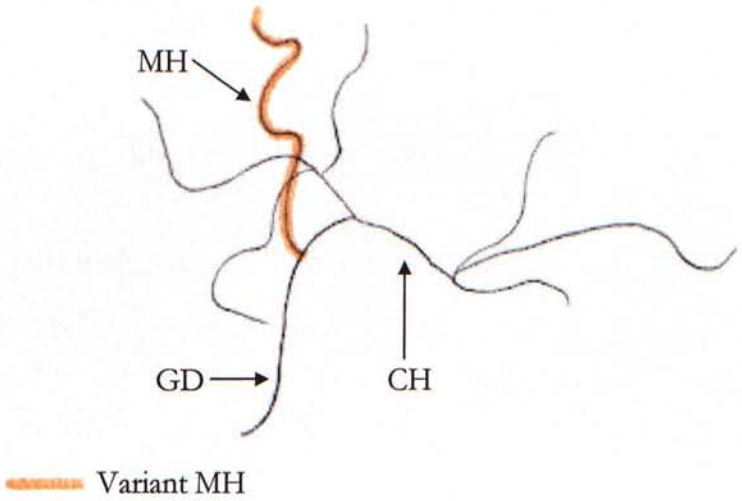


Figure 6.24b Schematic diagram showing 'MH fr GD'



6.6 Left hepatic artery

The left hepatic artery arises from the proper hepatic artery classically (figure 6.04 & figure 6.05). In this study, 77.5% of the subjects have this classical presentation of the left hepatic artery. Other observed variants of the origin of the left hepatic artery are the common hepatic artery, left gastric artery, and coeliac axis. Occasionally there is an accessory LH from the left gastric artery and sometimes the left hepatic artery is absent. The different percentages are tabulated in table 6.06 and figure 6.26.

Table 6.06 Origin of the left hepatic artery

Vessel origin	Number observed	Percentage	Illustration
Proper hepatic artery	214	77.5%	figure 6.04, 6.05
Common hepatic artery	40	14.5%	figure 6.27
Left gastric artery	20	7.2%	figure 6.28
Accessory from LG	11	4.0%	figure 6.29
Coeliac axis	1	0.4%	figure 6.16a, 6.16b
Absent	1	0.4%	figure 6.15a

Figure 6.26 Origin of the left hepatic artery

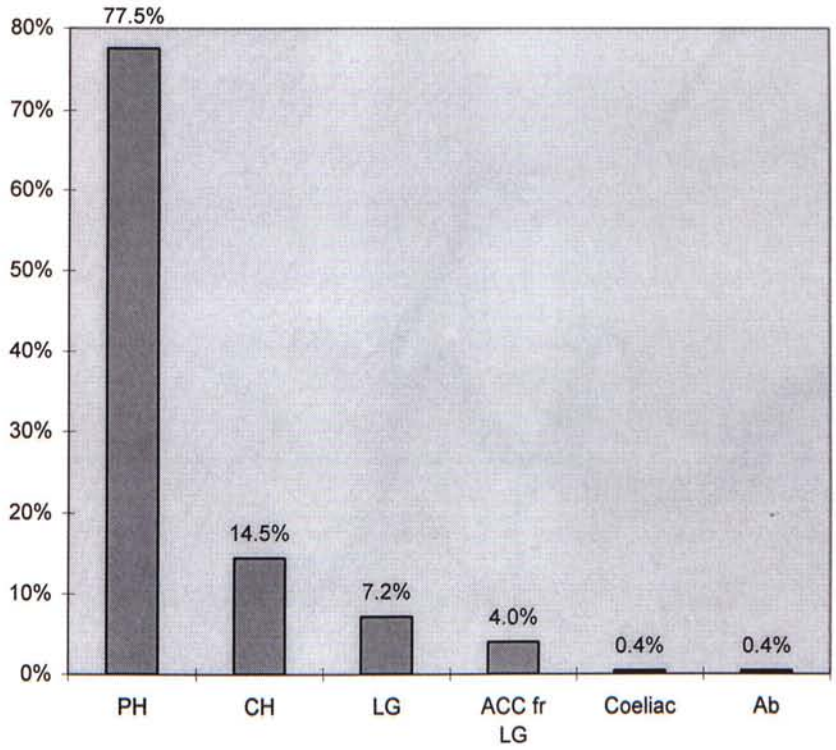


Figure 6.27a LH fr CH

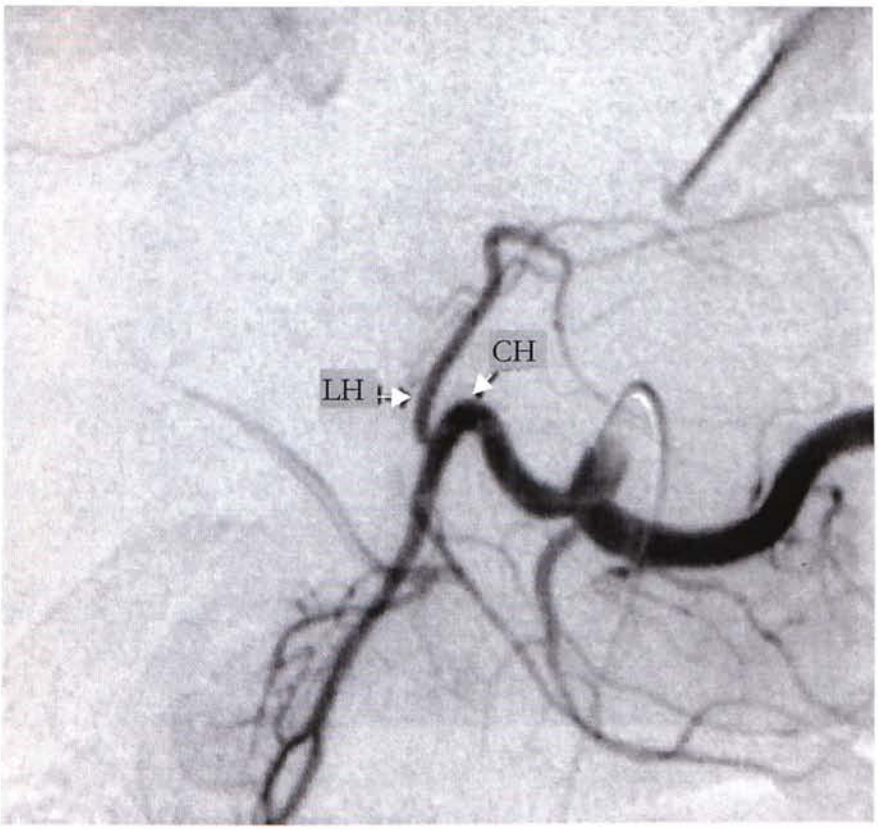
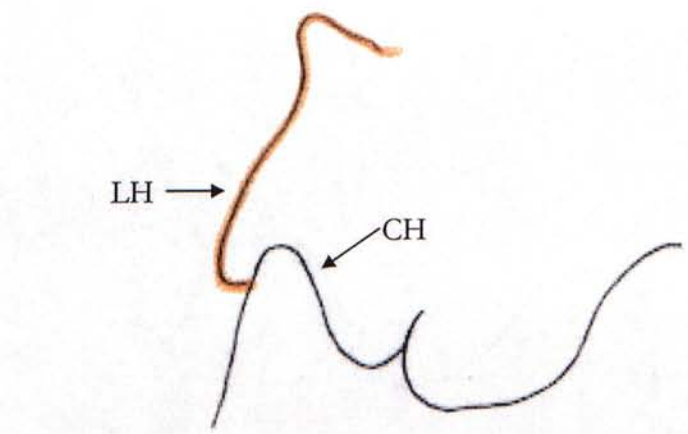


Figure 6.27b Schematic diagram showing 'LH fr CH'



Variant LH

Figure 6.28a LH fr LG

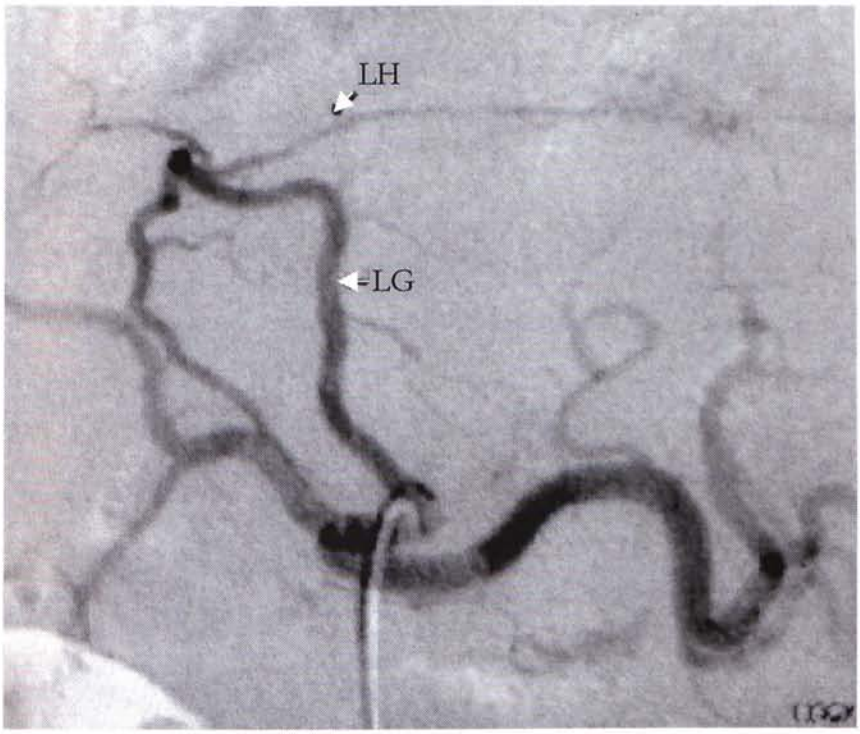


Figure 6.28b Schematic diagram showing 'LH fr LG'

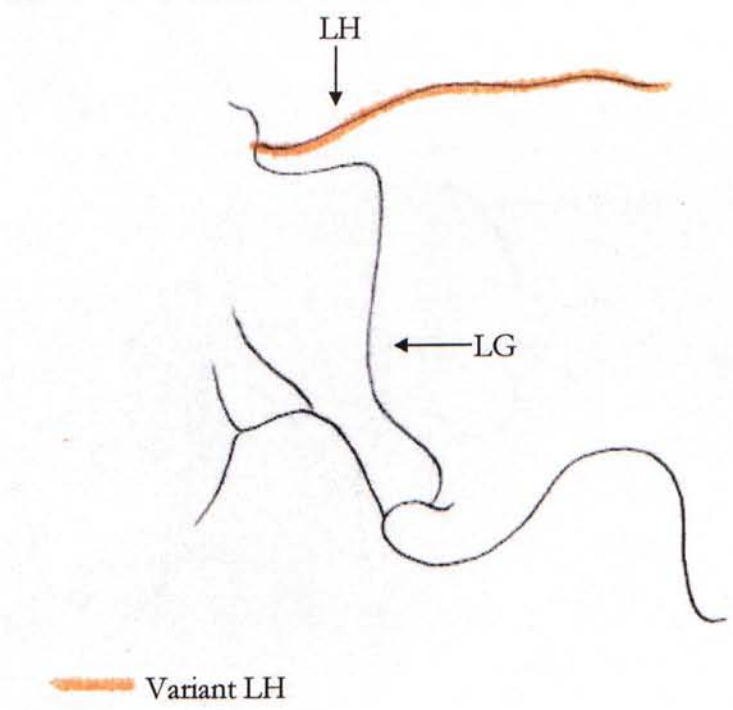


Figure 6.29a ACC LH fr LG

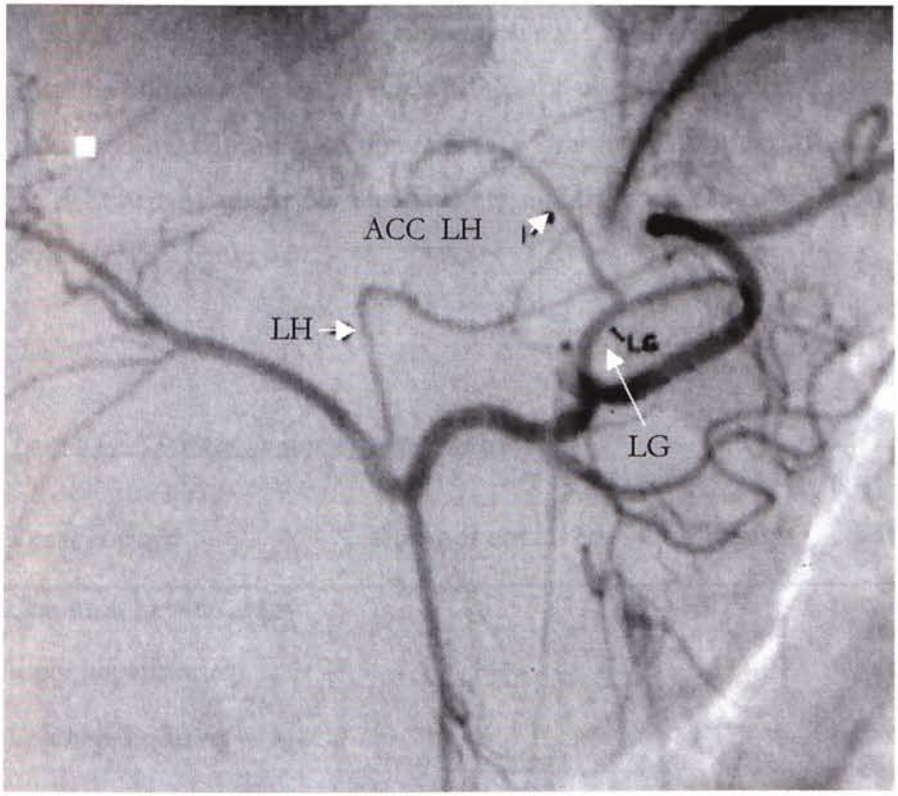
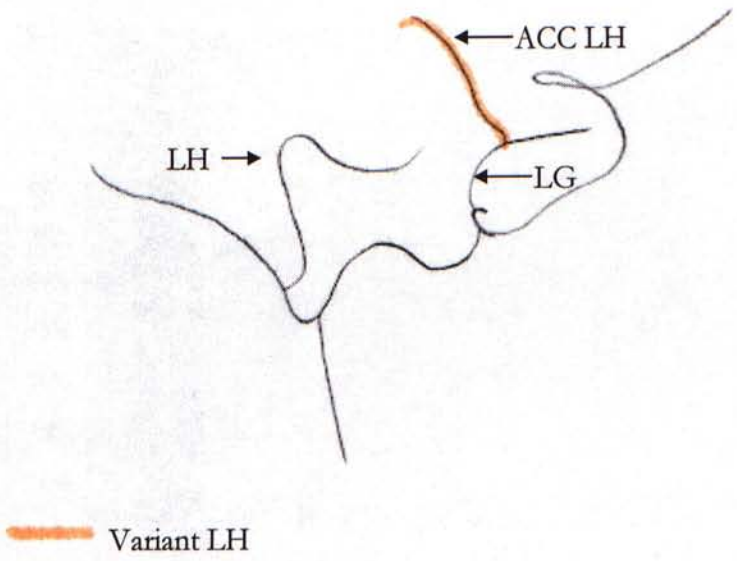


Figure 6.29b Schematic diagram showing 'ACC LH fr LG'



6.7 Gastroduodenal artery

In this study, 93.1% of the subjects have the gastroduodenal artery originating from the common hepatic artery which is the classical presentation of this vessel (illustrated by figure 6.04 & figure 6.05). Other variant origins of this vessel are the right hepatic artery, left hepatic artery, and the coeliac axis. These are listed in table 6.07 and figure 6.31.

Table 6.07 Origin of the gastroduodenal artery

Vessel origin	Number observed	Percentage	Illustration
Common hepatic artery	257	93.1%	figure 6.04, 6.05
Right hepatic artery	14	5.1%	figure 6.32
Left hepatic artery	4	1.4%	figure 6.33a, 6.33b
Coelaic axis	1	0.4%	figure 6.16a, 6.16b

Figure 6.31 Origin of the gastroduodenal artery

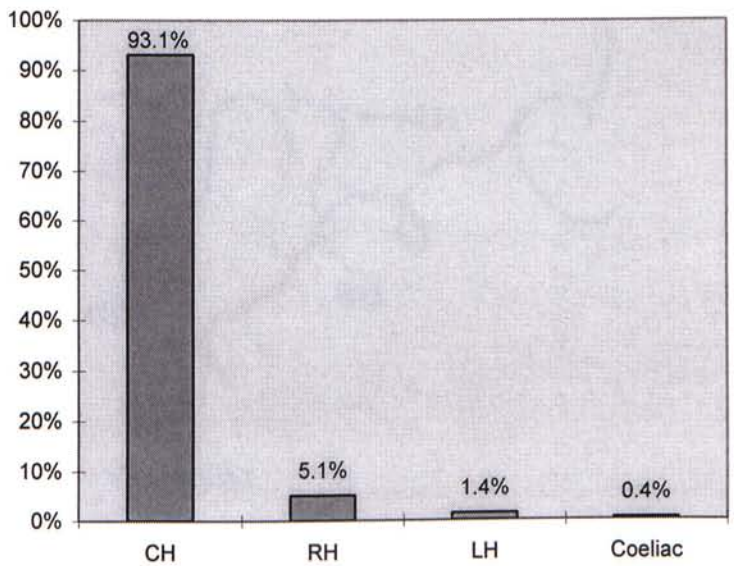


Figure 6.32a GD fr RH

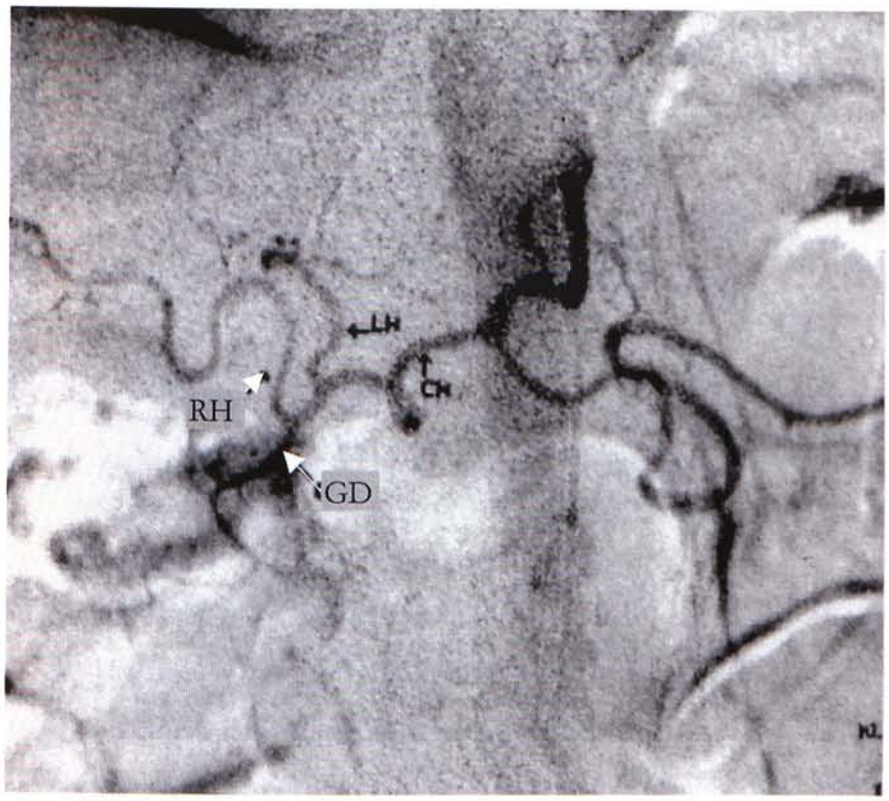


Figure 6.32b Schematic diagram showing 'GD fr RH'

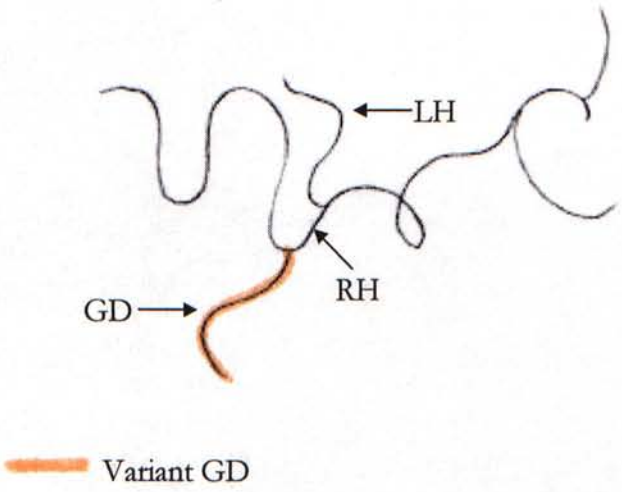


Figure 6.33a1 GD fr LH

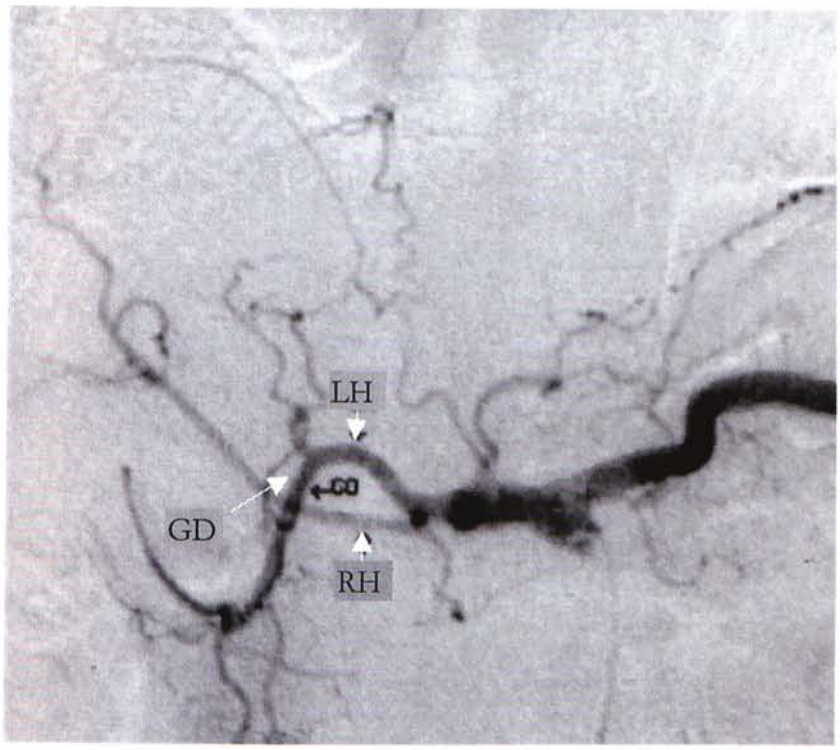


Figure 6.33a2 Schematic diagram showing 'GD fr LH'

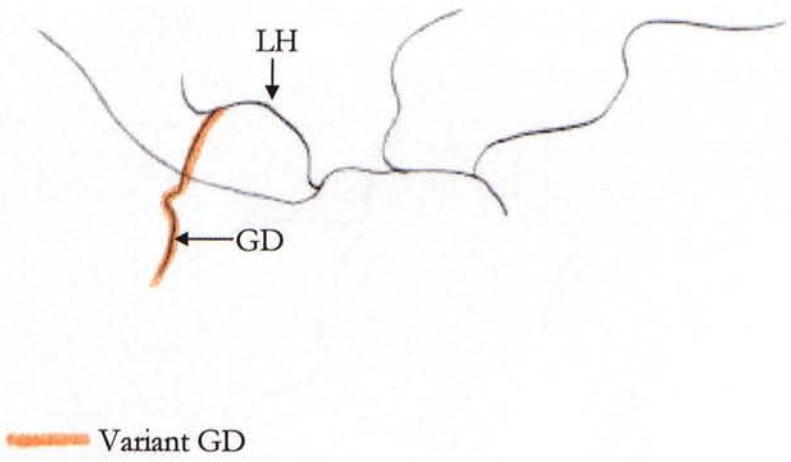


Figure 6.33b1 GD fr LH

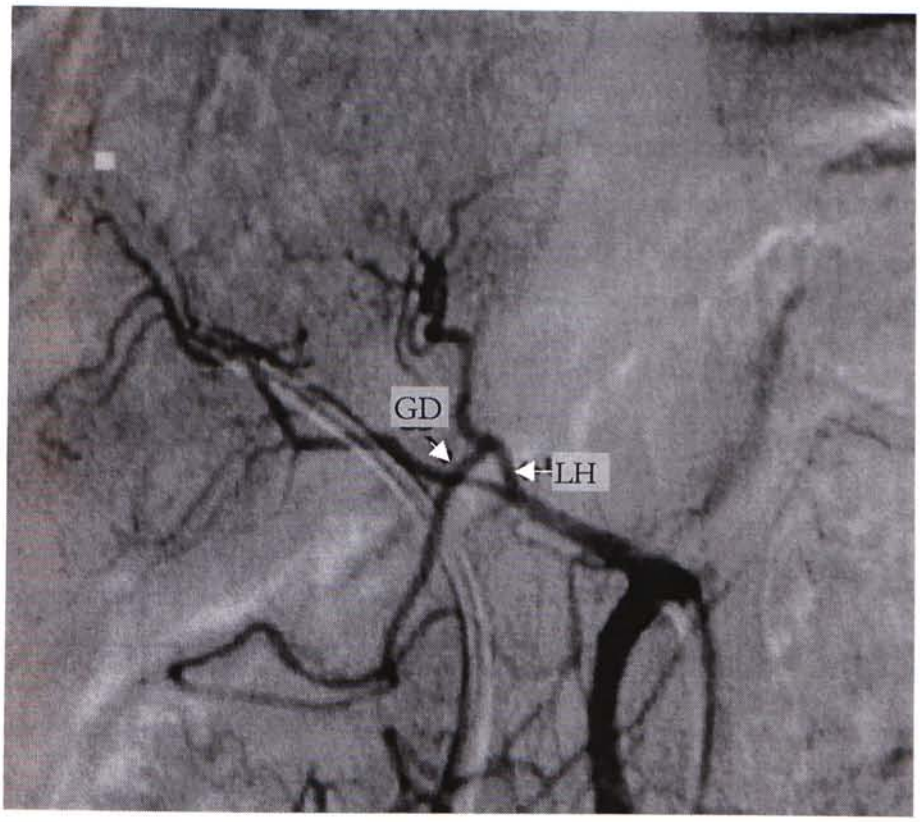
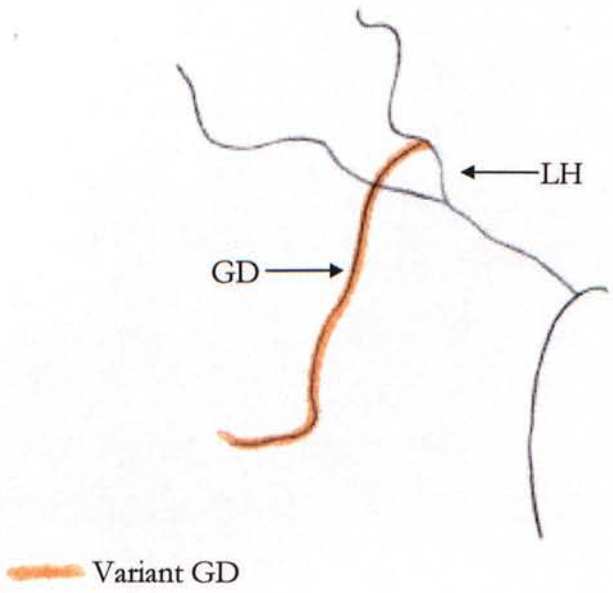


Figure 6.33b2 Schematic diagram showing 'GD fr LH'



6.8 Right gastric artery

From anatomy text books, the right gastric artery originates from the proper hepatic artery (figure 6.04 & figure 6.05). Although this artery is usually small, it has many variants and all these variants warrant special attention when considering Selective Internal Radiation (SIR) treatment for hepatocellular carcinoma. Table 6.08 and figure 6.34 list the origins and the respective percentages observed.

Table 6.08 Origin of the right gastric artery

Vessel origin	Number observed	Percentage	Illustration
Proper hepatic artery	116	42.0%	figure 6.04, 6.05
Left hepatic artery	98	35.5%	figure 6.35a, 6.35b
Right hepatic artery	28	10.1%	figure 6.36a, 6.36b
Common hepatic artery	12	4.3%	figure 6.37a, 6.37b
Gastroduodenal artery	10	3.6%	figure 6.38a, 6.38b
Absent	6	2.2%	
Middle hepatic artery	3	1.1%	figure 6.23
Bifurcation of PH	2	0.7%	figure 6.39a, 6.39b
Bifurcation of CH	1	0.4%	figure 6.40a, 6.40b

Figure 6.34 Origin of the right gastric artery

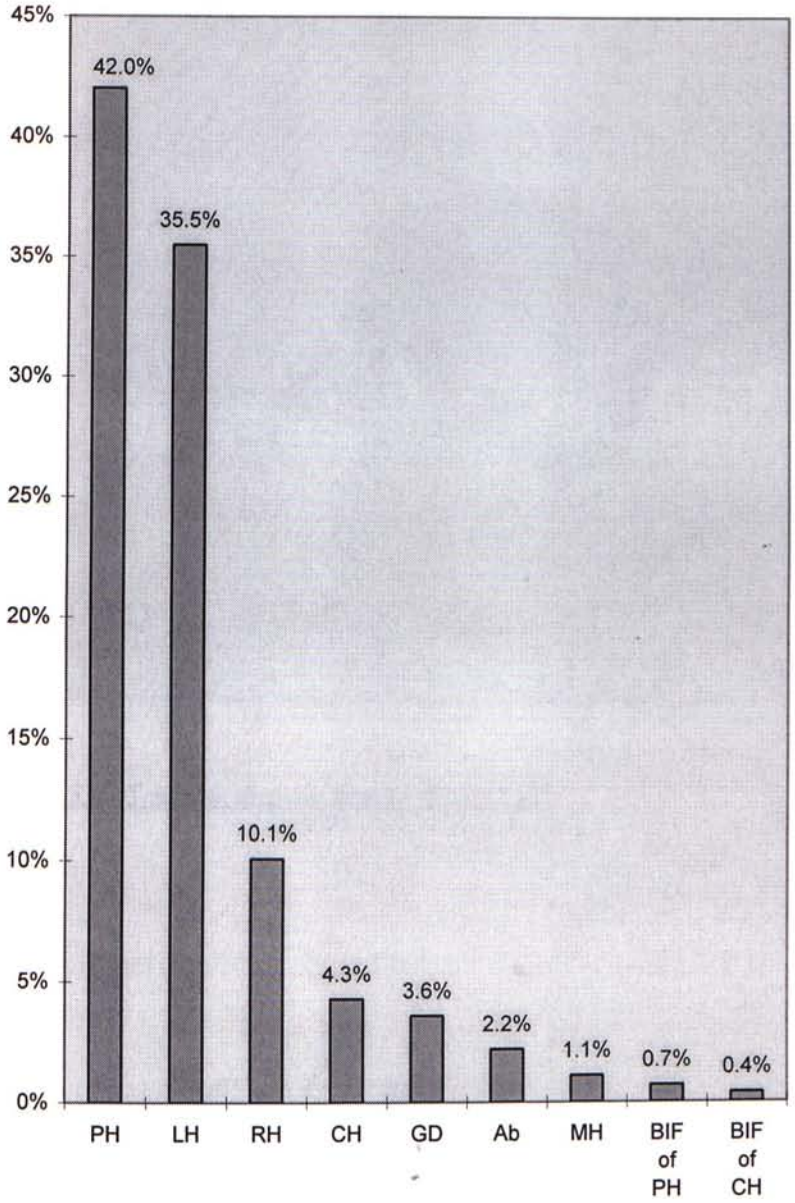


Figure 6.35a RG fr LH

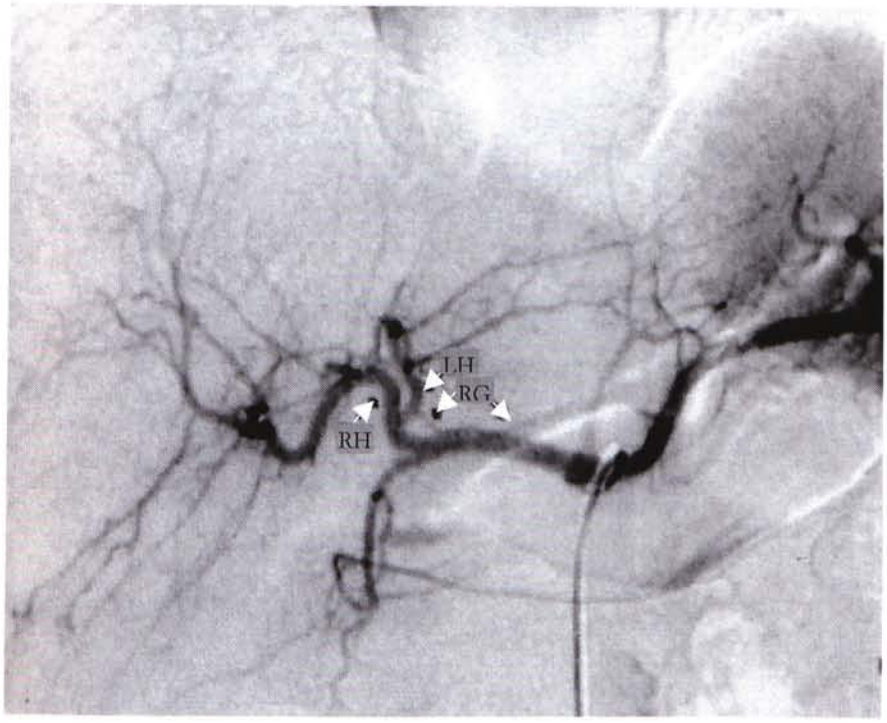
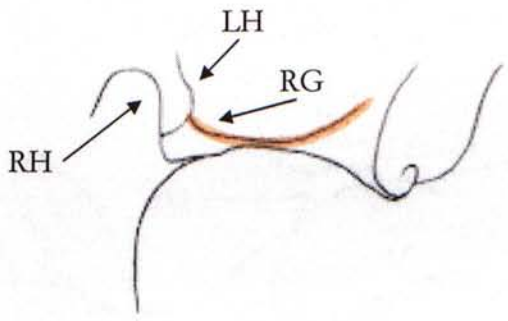


Figure 6.35b Schematic diagram showing 'RG fr LH'



Variant RG

Figure 6.36a RG fr RH

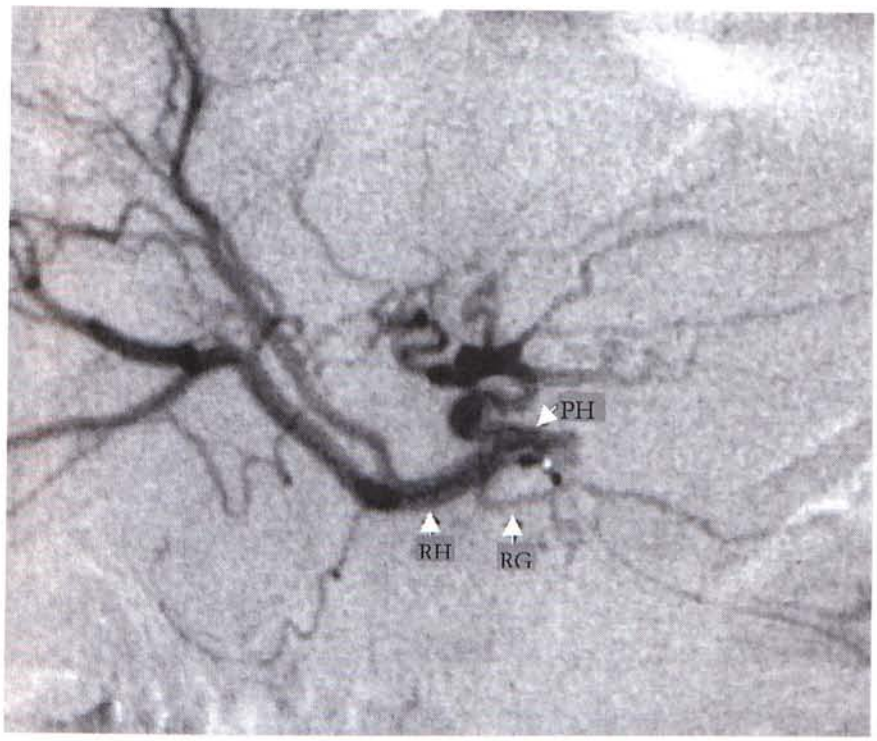
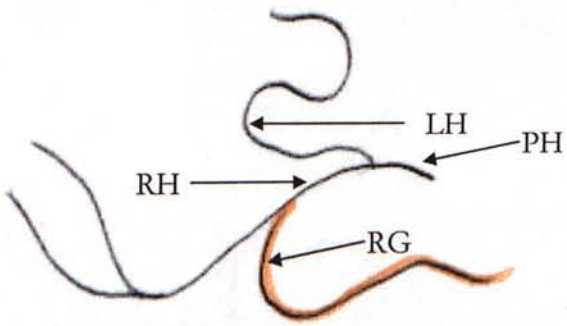


Figure 6.36b Schematic diagram showing 'RG fr RH'



Variant RG

Figure 6.37a RG fr CH

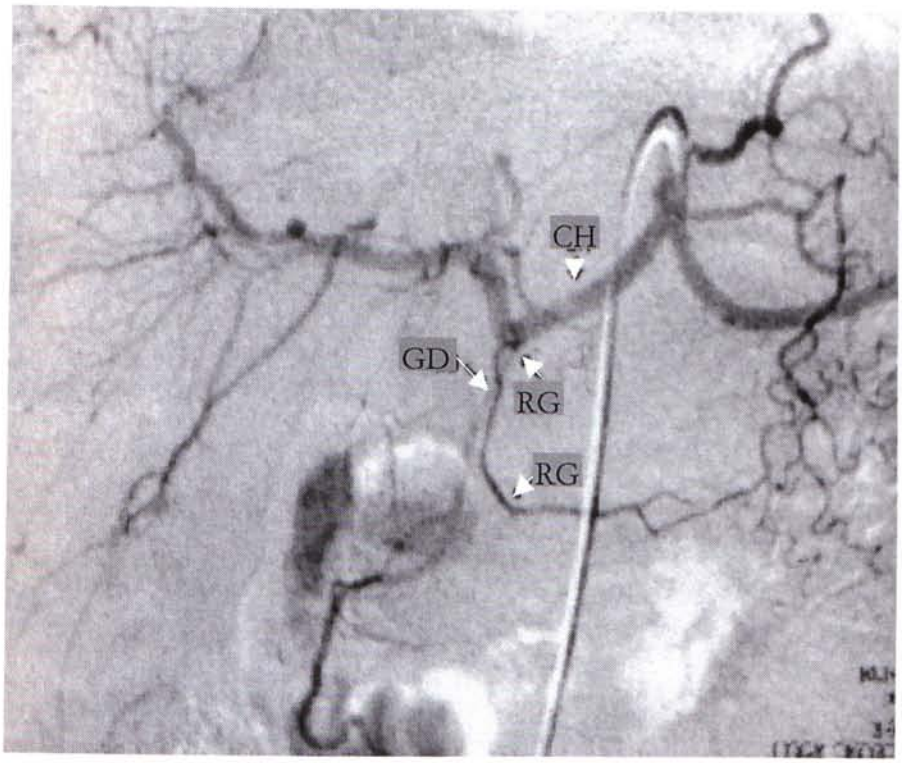


Figure 6.37b Schematic diagram showing 'RG fr CH'

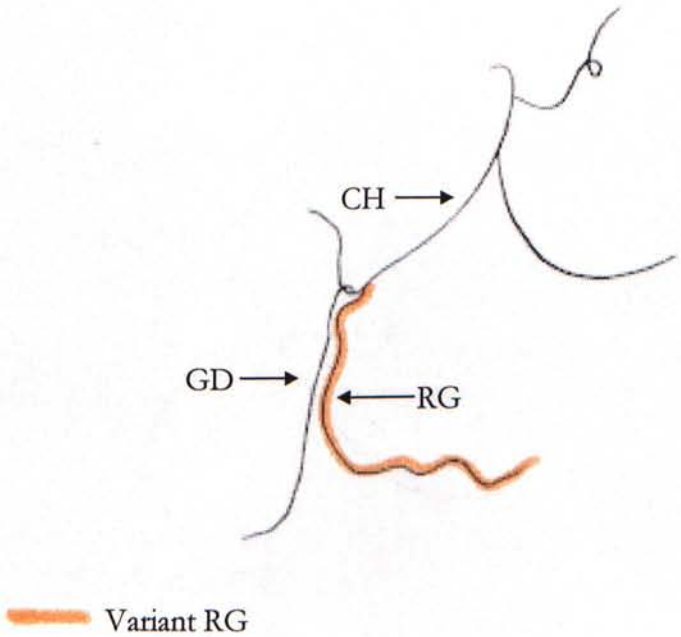


Figure 6.38a RG fr GD

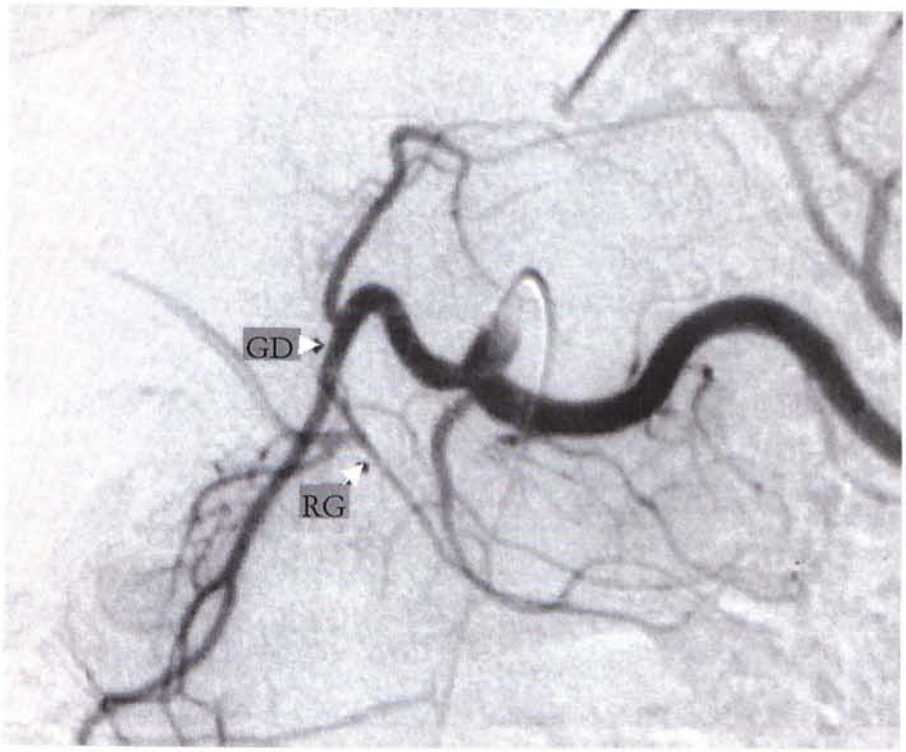


Figure 6.38b Schematic diagram showing 'RG fr GD'

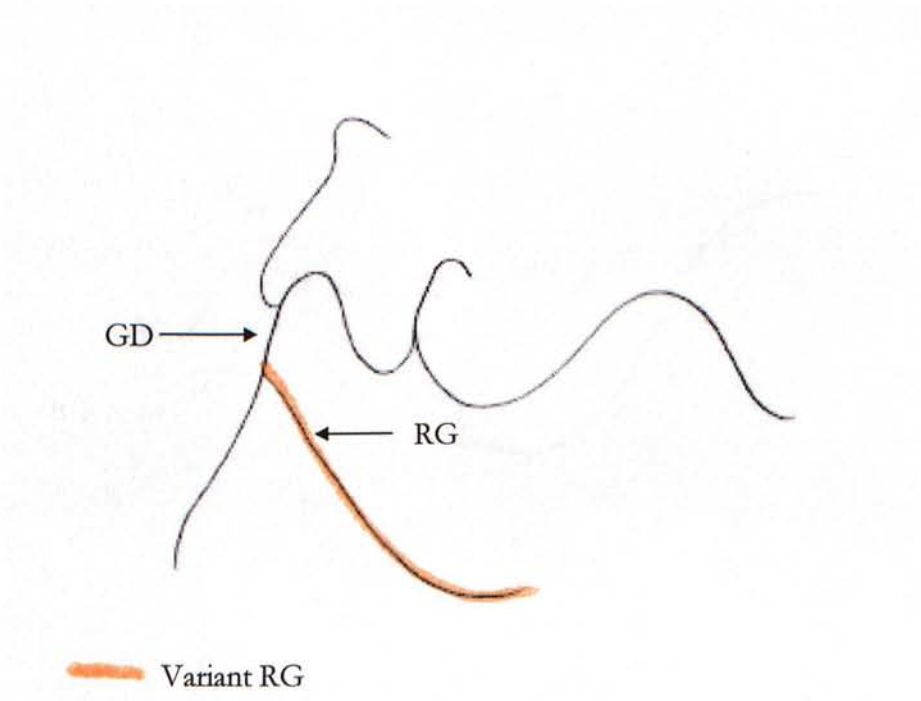


Figure 6.39a RG fr Bifurcation of PH

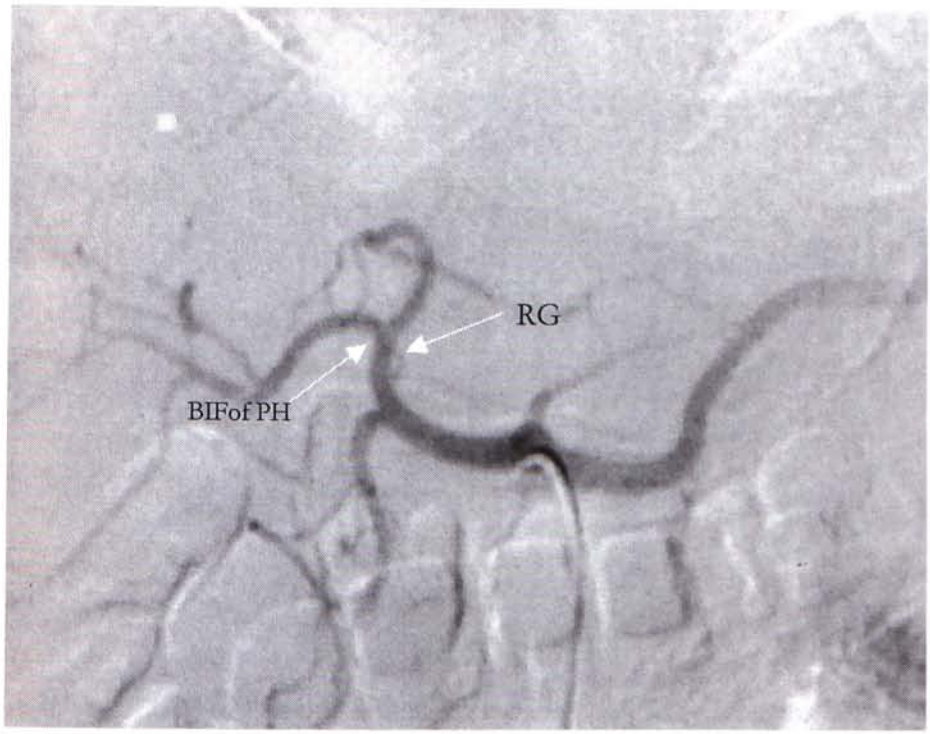


Figure 6.39b Schematic diagram showing 'RG fr Bifurcation of PH'

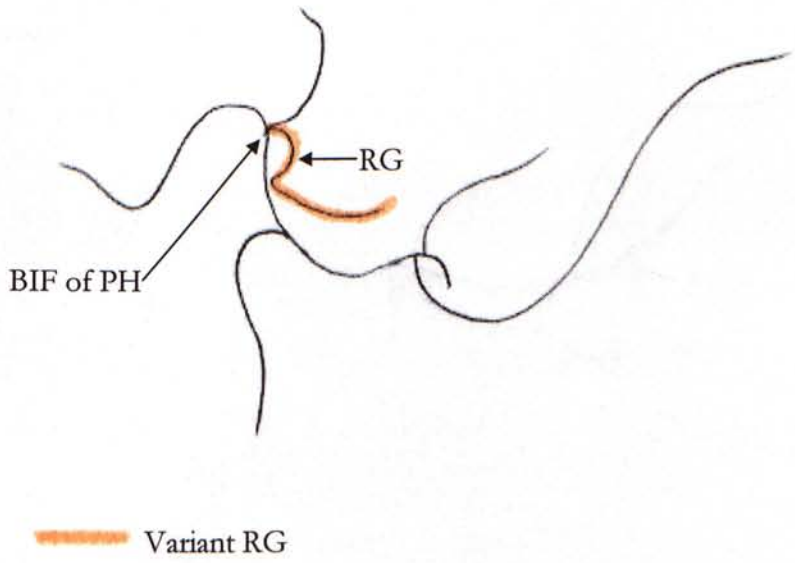


Figure 6.40a RG fr Bifurcation of CH

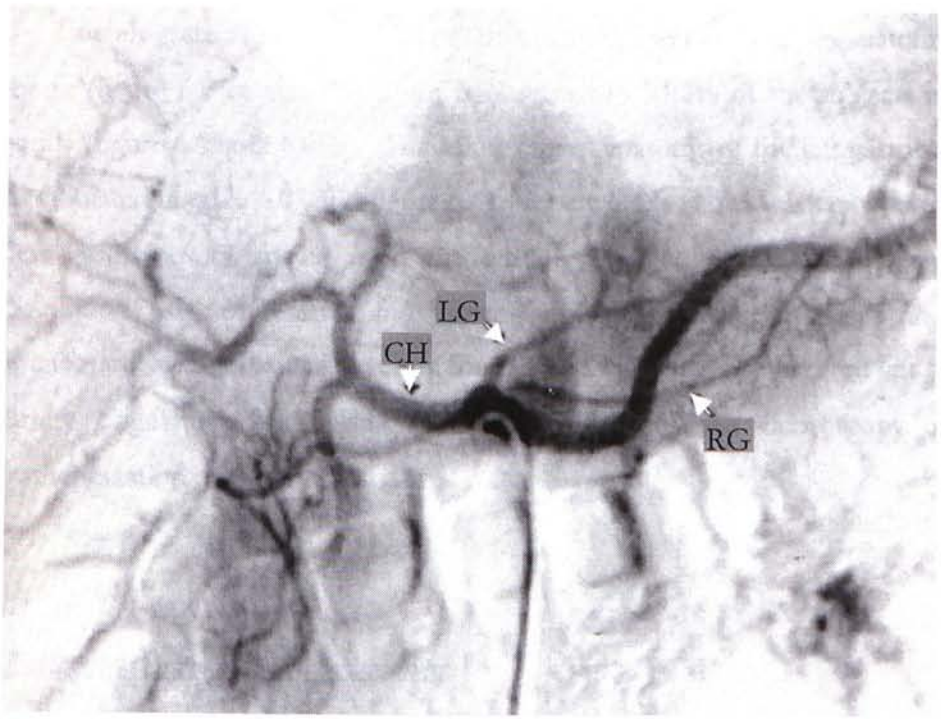
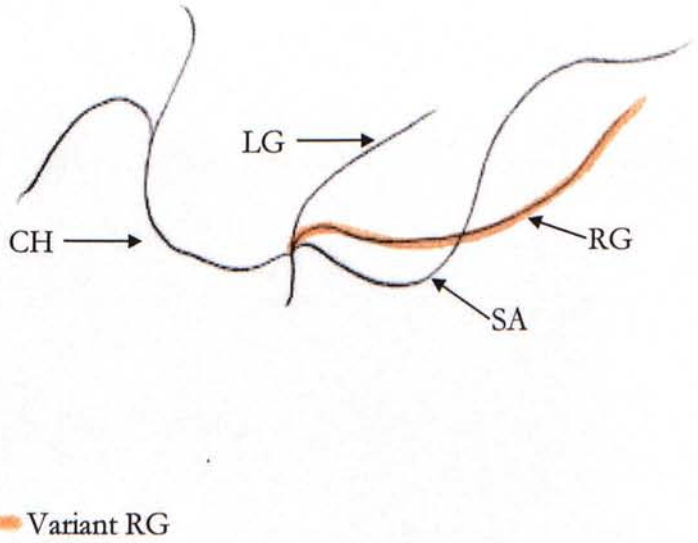


Figure 6.40b Schematic diagram showing 'RG fr Bifurcation of CH'



6.9 Left gastric artery

The left gastric artery arises from the coeliac axis as quoted from anatomy text book (figure 6.04a) and this norm is observed in 94.2% of the subjects in this study (figure 6.05a & 6.05b). The other variant anatomy of the left gastric artery is tabulated in table 6.09 and figure 6.41. Figure 6.42a shows a lateral view of the left gastric artery arising from the aorta. This radiograph is copied from Saadoon's Atlas of Normal and Variant angiographic anatomy, since no extra angiograms were obtained specially for this study and the six observed left gastric artery originating from aorta were observed from test fluoroscopy during catheterization of the coeliac axis.

Table 6.09 Origin of the left gastric artery

Vessel origin	Number observed	Percentage	Illustration
Coeliac	261	94.6%	figure 6.05a, 6.05b
Aorta	6	2.2%	figure 6.42a, 6.42b
Bifurcation of Coelaic	5	1.8%	figure 6.43
Absent	4	1.4%	

Figure 6.41 Origin of the left gastric artery

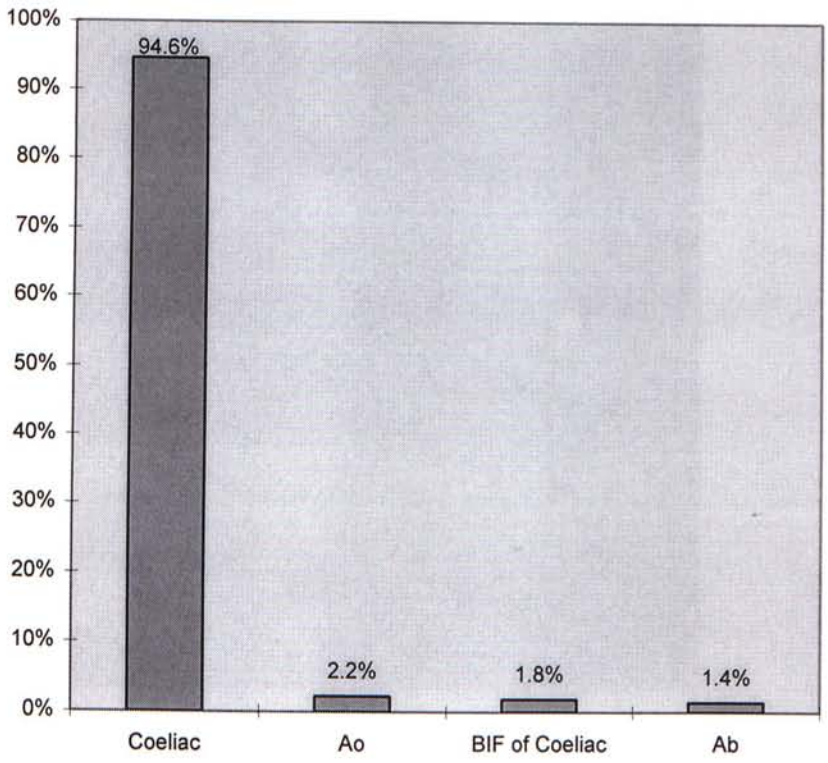


Figure 6.42a LG fr Ao (Lateral view)
(From Atlas of Normal and Variant Angiographic Anatomy by Saadoon Kadir)

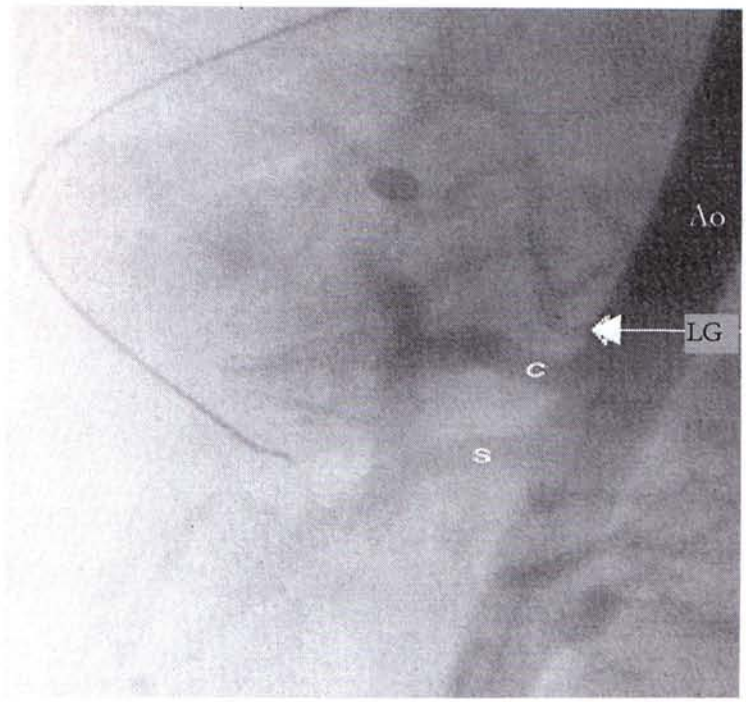


Figure 6.42b Schematic diagram showing 'LG fr Ao (Lateral view)'

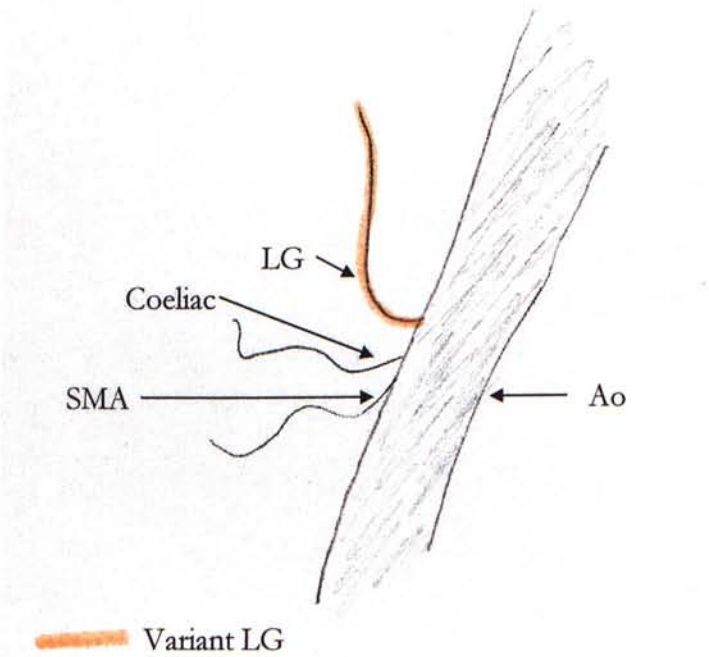
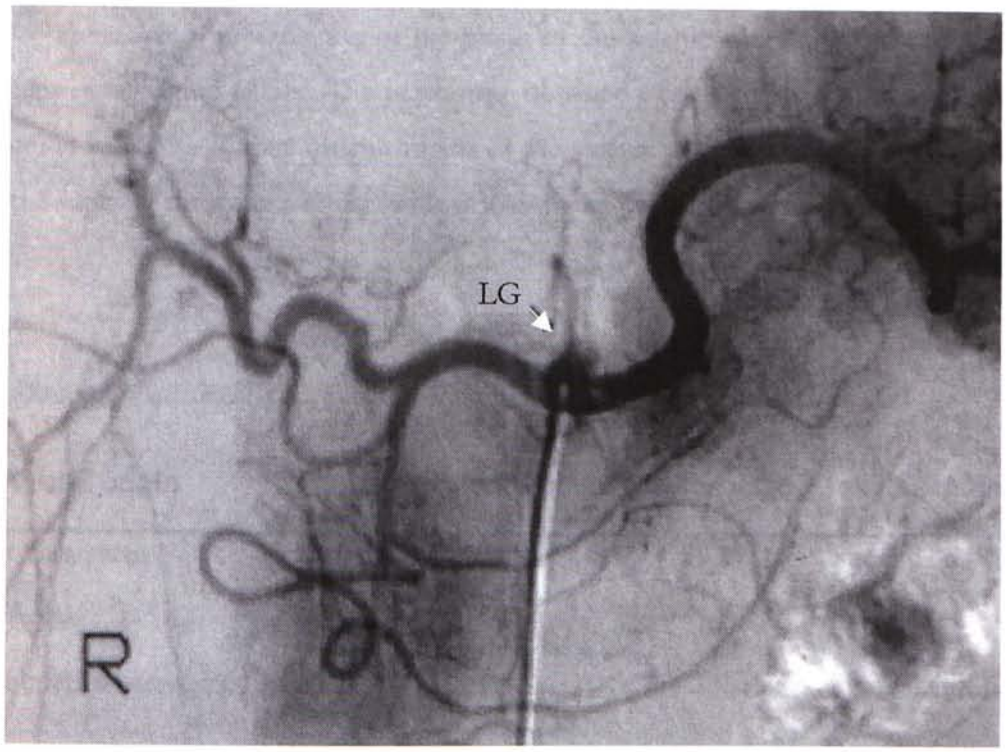


Figure 6.43 LG fr BIF of Coeliac



6.10 Splenic artery

The classical presentation of the origin of the splenic artery is shown here in figures 6.05a and 6.05b. The percentage obtained of this classical pattern in this study is 99.3%. Other variant origins of the splenic artery include the aorta and the superior mesenteric artery (table 6.10 & figure 6.45).

Table 6.10 Origin of the spenic artery

Vessel origin	Number observed	Percentage	Illustration
Coelaic axis	274	99.3%	figure 6.05a, 6.05b
Aorta	1	0.4%	figure 6.08b
Superior mesenteric artery	1	0.4%	figure 6.46a, 6.46b

Figure 6.45 Origin of the splenic artery

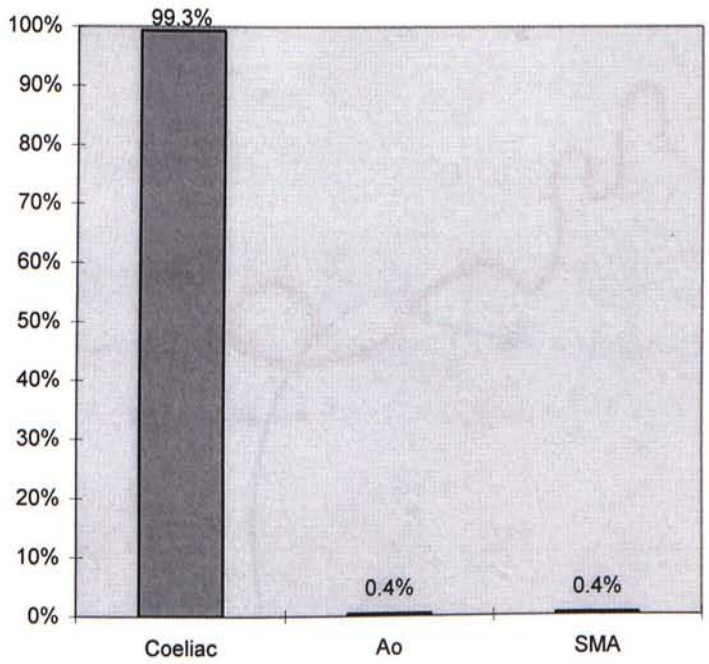


Figure 6.46a SA fr SMA

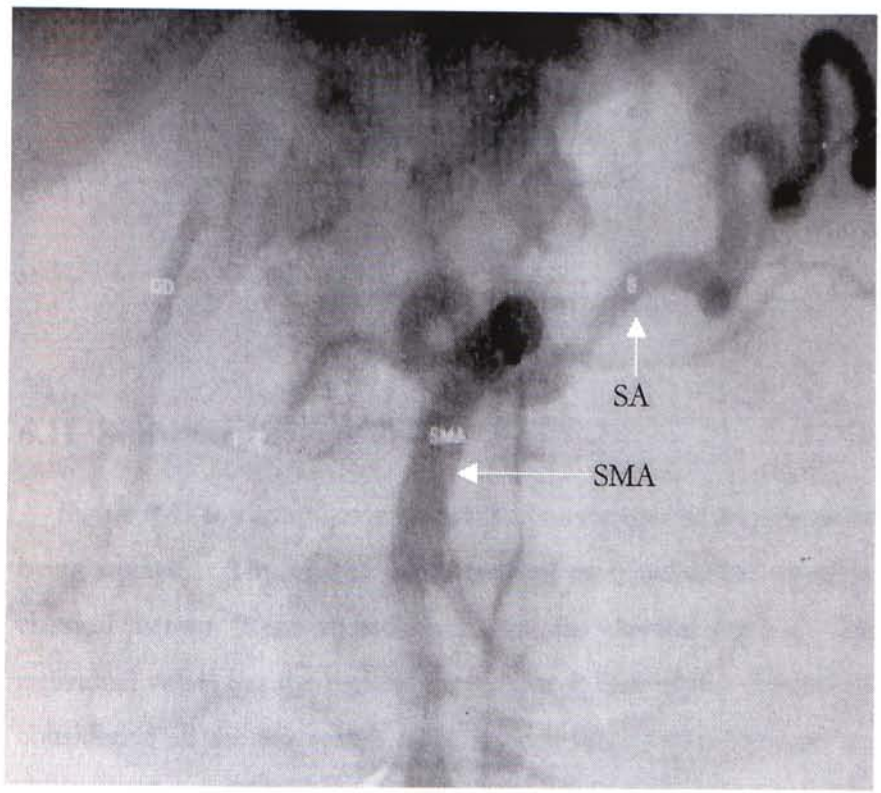
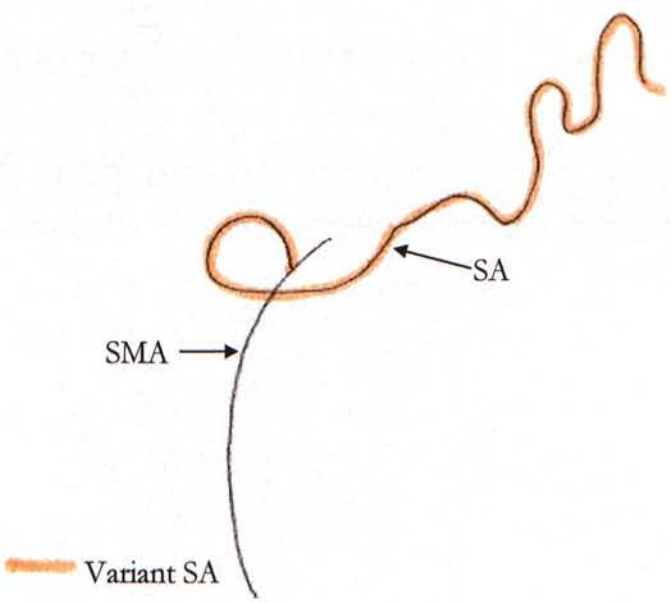


Figure 5.46b Schematic diagram showing 'SA fr SMA'



6.11 Summary of results

Figure 6.47 is a summary chart of the percentages of the origins of the vessels being studied. The highest occurrence of each individual vessel is that of the classical pattern. (Refer to section 3.1 for the classical pattern) Although each individual vessel has the highest occurrence as that of the classical pattern, when considering all the ten vessels, only 28% of the subjects have no variant as that shown in figure 6.04a. In another words, 72% of the subjects have at least one variant (figure 6.48). When considering the hepatic arterial blood supply only, the varied character of the arterial blood supply to the liver are due primarily to the varied origins of the three major liver arteries, viz., the right, the left and the middle hepatics (Michels,1955). There are 55.4% of the subjects possessing all the three liver arteries with the classical pattern (Figure 6.49). Michels obtained a 55% in his study. These 2 percentages are tested for significance of difference using the Chi-square test. The p-value obtained is 0.47 which is greater than 0.05. This result do not differ significantly from that obtained by Michels.

Figure 6.47 Summary chart of the percentages of the vessel origins

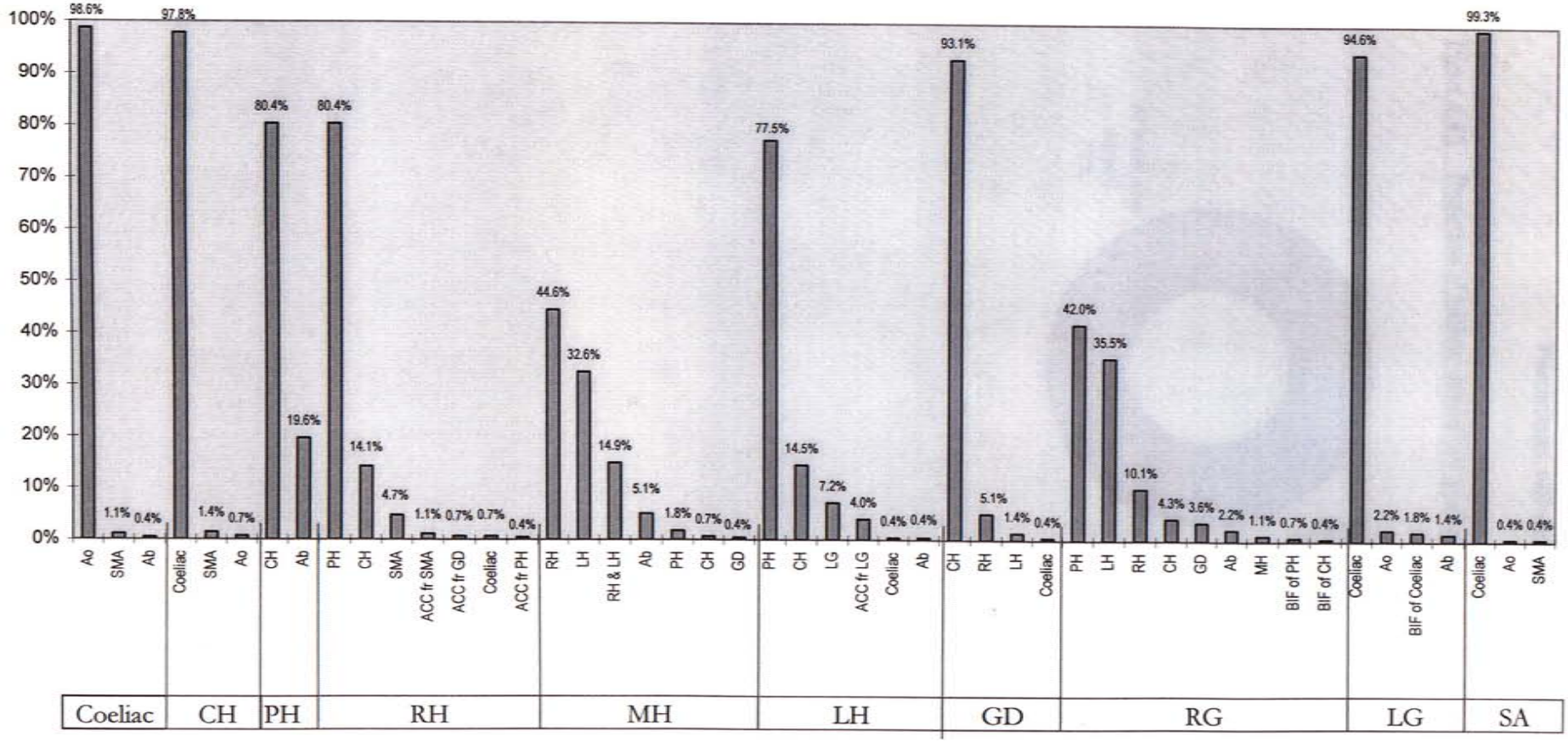


Figure 6.48 Proportion of 'without variant' & 'with at least one variant'

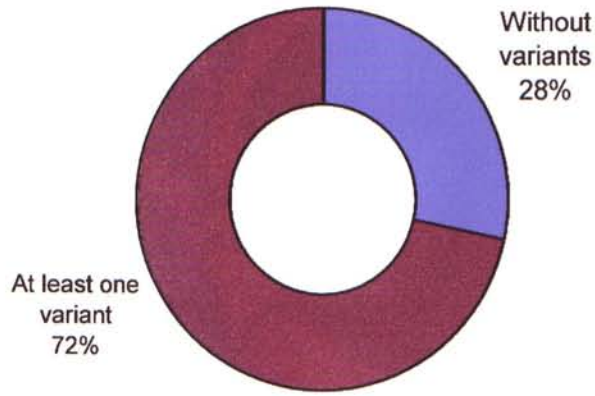
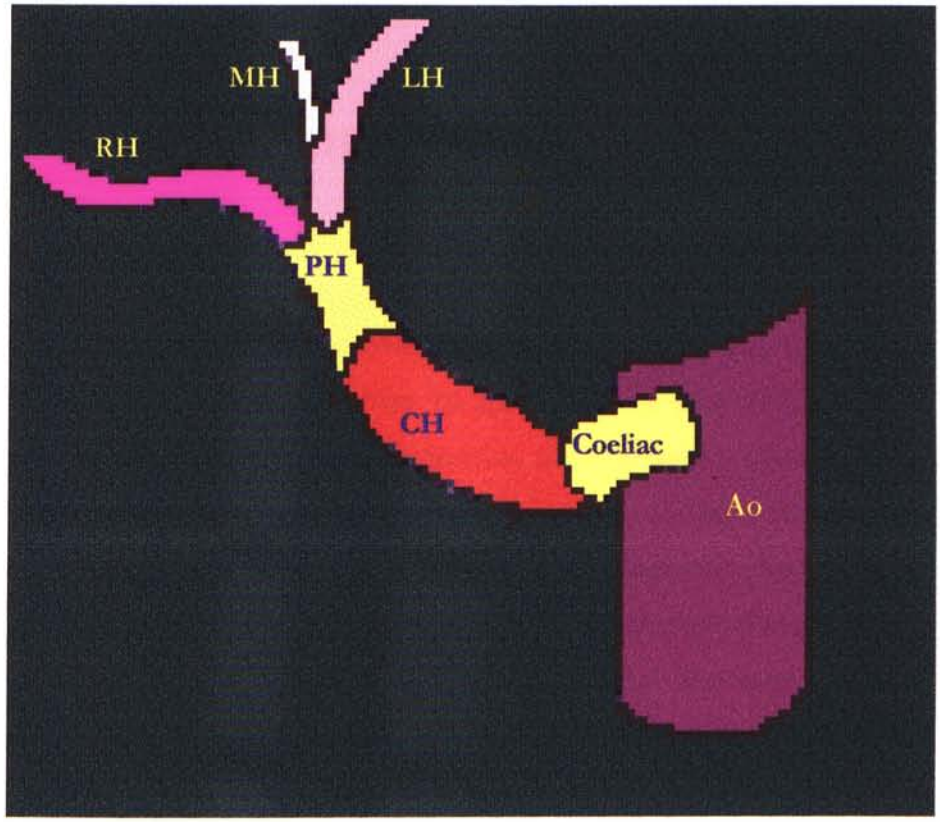


Figure 6.49 A 'normal' arterial supply to the liver



Chapter 7

DISCUSSION

7.1 Introduction

For surgeons, the best way to avoid injury to blood vessels is to know them, and to know how, when and **where** to ligate them properly (Michels,1955). Whereas, for the radiological interventionalist, the only way to achieve precise endovascular treatment is to know the vascular pathways and their final capillary beds, and to know how, **where** and how much of the therapeutic drug to deliver properly. The objective of ^{90}Y-SIR is to deliver the maximum radiation dose to the tumour avoiding any dose to significantly sensitive 'innocent bystander' arteries. In the situation under consideration, these are the arteries supplying the stomach and the duodenum. The endovascular treatment of Selective Internal Radiation using ^{90}Y for hepatocellular carcinoma is described in chapter two, section 2.7. The **'where'** for both the surgeon and the interventionist closely associates with the theme of this study, anomalies of vessels. In this study, the anomalies of the coeliac axis and its branches are studied. The previous large scale study of the arterial variants of the upper abdomen was done in 1955 by Michels. Whence, 200 cadavers were dissected and analysed. In this study, 276 subjects' arteriograms of the coeliac axis and superior mesenteric artery were analysed. The percentages of the variants of the coeliac axis and its branches in Hong Kong Chinese were established and presented in the chapter six.

In the following section, endovascular treatment procedure is classified into two main types with four subtypes. The method of approach in the classical pattern of the coeliac axis is described. Each of the ten vessels studied are discussed in turn with regard to the following aspects:

- Comparison with Michels' study.
- The influence of the anomalies upon intraarterial ^{90}Y treatment for HCC.

Finally there are three sections on :

1. comparison of the results of this study with that quoted by Michels,
2. comparison of the findings in the group of subjects with hepatocellular carcinoma with that in the group without hepatocellular carcinoma,
3. and, comparison of the results obtained from the male subjects and that from the female subjects.

7.2 Selective Internal RadiationTherapy

Results from Lau & Ho's (1997) study show that intraarterial ^{90}Y microspheres given through an angiographic route is a feasible treatment for non-resectable HCC. During the angiographic procedure for infusion of ^{90}Y microspheres, the two major objectives are, first, to deliver the ^{90}Y microspheres to the HCC and, second, to avoid radiation induced gastritis due to this treatment.

The first objective is achieved by delivering the ^{90}Y microspheres superselectively to the arterial supply of the tumour.

The measures taken to avoid radiation induced gastritis include the followings :

1. Superselective catheterization of the hepatic artery by-passing the gastroduodenal artery and the right gastric artery for the ^{90}Y microspheres infusion.

2. The ^{90}Y microspheres suspension is rendered visible fluoroscopically by adding to it a radiopaque contrast medium, Omnipaque 300.

3. Infusion of the ^{90}Y microspheres is performed under fluoroscopic monitoring, thus preventing reflux of the radioactivity into the gastroduodenal artery.

With the two mentioned objectives in mind, basically, the endovascular treatment procedure can be classified into two types as follows :

- Type I

  Superselective catheterization of the hepatic arteries.

- Type II

  Superselective catheterization of the hepatic arteries preceded by embolization of some relevant arteries either to protect the stomach or for flow re-distribution.

Each type can be further subdivided into the following subtypes:

- Type I-a

  Superselective catheterization of one hepatic artery and infusing it with the ^{90}Y microspheres.

- Type I-b

  Superselective catheterization of more than one hepatic arteries and infusing them with ^{90}Y microspheres in turn.

- Type II-a

  Superselective catheterization of one or more hepatic arteries and infusing them in turn with ^{90}Y microspheres preceded by embolization of one or more gastric arteries to prevent radioactive microspheres going to the stomach and duodenum. Embolization of the gastric arteries causes no harm to the gastro-intestinal tract as the other gastric vessels will hypertrophy to compensate for the embolized artery/arteries.

- Type II-b

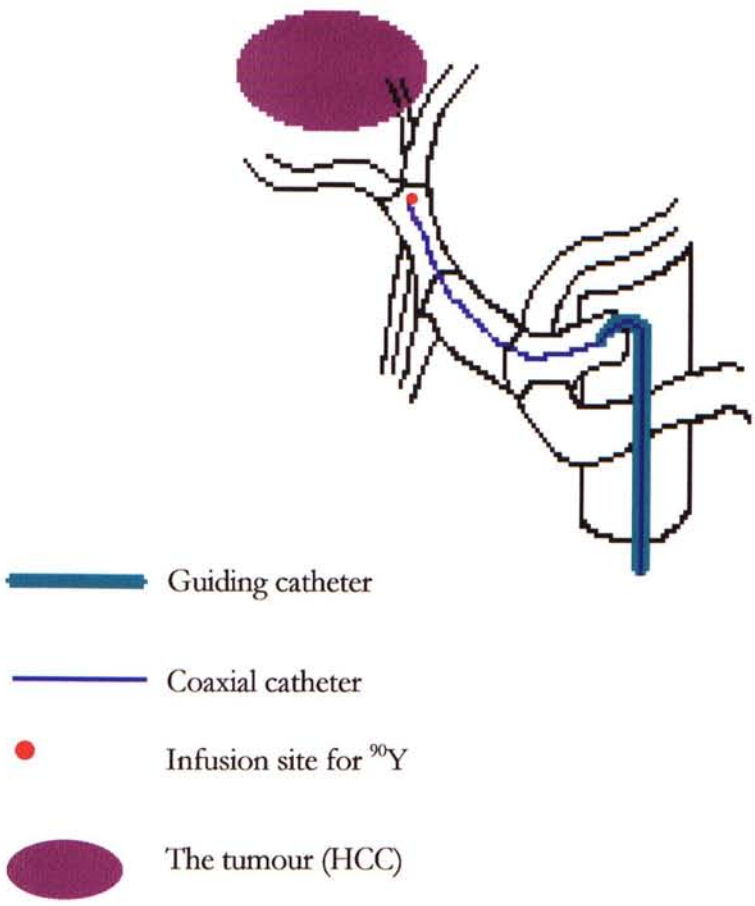
  Superselective catheterization of one or more hepatic arteries and infusing them in turn with ^{90}Y microspheres preceded by embolization of a hepatic artery, which may or may not be a variant, for flow re-distribution. The embolization site is to be at the proximal end of the artery and collaterals will open virtually instantaneously. This collateral circulation will carry more ^{90}Y microspheres to the tumour.

No matter which type of endovascular treatment procedure is employed, the guiding catheter should be placed at the relatively larger vessel such as the coeliac axis or the common hepatic artery. A coaxial catheter threaded inside the guiding

catheter and advanced to the desired site is used for infusion of the ^{90}Y microspheres or for delivering the embolization agent.

Figure 7.01 shows a hypothetical HCC involving both the right and the left lobes, and the appropriate catheter placement is shown. The type I-a endovascular treatment procedure is applied. The tip of the guiding catheter is placed at the proximal end of the coeliac axis and the tip of the coaxial catheter is placed at the proper hepatic artery by-passing the right gastric artery. In this way, the ^{90}Y microspheres can be delivered to the tumour and radiation induced gastritis can be avoided.

Figure 7.01 Catheter placement for ^{90}Y delivery for the classical pattern of the coeliac axis

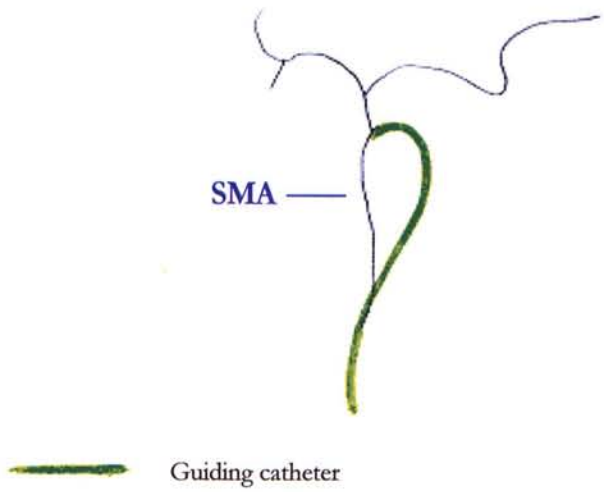


7.3 The coeliac axis

In this study, no direct comparison with Michels' can be made since the classification method is different. When considering catheter placement for SIR treatment, for the majority of the subjects who have the coeliac axis originating from the aorta, the guiding catheter should be advanced through the abdominal aorta into the proximal part of coeliac axis, as shown in figure 7.01. If the coeliac axis arises from superior mesenteric artery, the guiding catheter should be advance through the abdominal aorta into the superior mesenteric artery (figure 7.02 & 6.07a). For the absent coeliac, the origin of the common hepatic artery has to be ascertained. As in the case shown in figure 6.08a, the common hepatic artery arises directly from the aorta, the guiding catheter should be advanced through the abdominal aorta and into the proximal end of the common hepatic artery (figure 7.03). The exact position of the coaxial catheter tip placement depends on the liver arterial supply, origin of the right gastric and gastroduodenal artery, and any other variant arterial supply of the liver and the stomach. These are discussed in the coming sections when appropriate.

Figure 7.02 Position of the guiding catheter for 'Coeliac fr SMA'

Trace drawing



From figure 6.07a

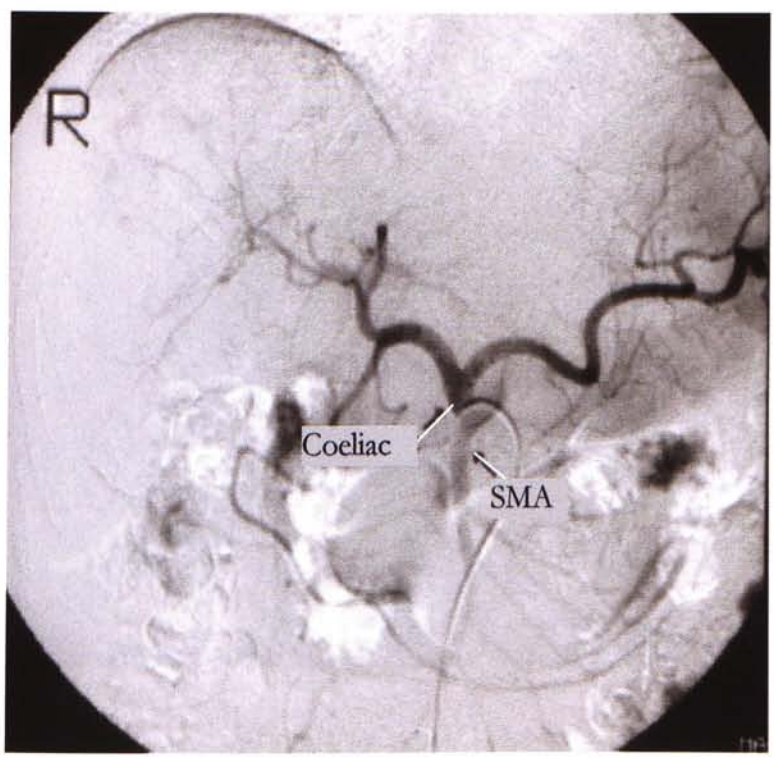
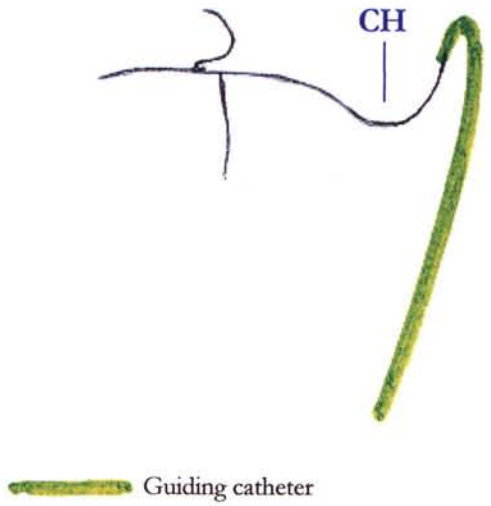
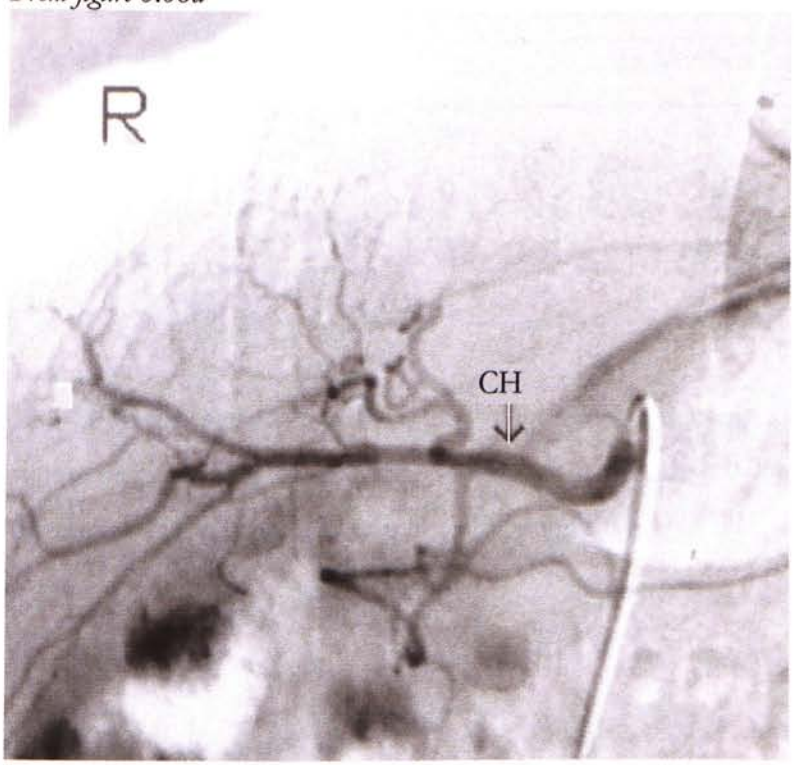


Figure 7.03 Position of the guiding catheter for 'CH fr Ao'

Trace drawing



From figure 6.08a



7.4 The common hepatic & proper hepatic artery

The proper hepatic artery is an important artery since in most cases the infusion site of the ^{90}Y microspheres is at the proper hepatic artery instead of at the common hepatic artery to avoid radiation induced gastritis. In Michels' work, the whole length of the hepatic vessel arising from the coeliac before branching into the right, left and middle hepatic arteries was termed the hepatic artery. Thus, during comparison, the hepatic artery referred in Michels' study could be analogous to the common hepatic artery in this study. Comparison of the percentages of the variant origins of the common hepatic artery in this study and Michels' is tabulated in table 7.01.

Table 7.01 Comparison of the % of the variant origins of the common hepatic artery

	Percentage		
Vessel origin	**In this study**	**In Michels' work**	**p-value**
Superior mesenteric artery	1.4%	2.5%	0.31
Aorta	0.7%	1.5%	0.35
Left gastric	0%	0.5%	0.42

The observed percentages from the two studies are compared using the Chi-square test. Each of the p-values obtained is greater than 0.05, which means that the results of the two studies do not differ significantly in this aspect.

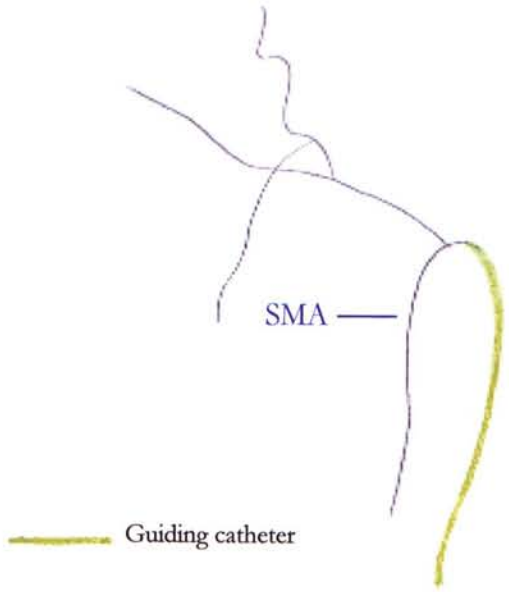
One significantly different result was obtained from Pedro Belou, of Buenos Aires (1915), in which there was a 6% of the bodies having the common hepatic artery arising from the superior mesenteric artery. (Michels, 1955) While in this study of the Hong Kong Chinese, a 1.4% is observed. This discrepancy may be due to racial difference or different research methodology or other reasons, but finding the reasoning behind is beyond the scope of this study.

When considering the catheter placement for SIR treatment for HCC, for the majority who have the common hepatic artery stemming from the coeliac axis, the coaxial catheter should be advanced out from the guiding catheter into the common hepatic artery (figure 7.01). For the variant of the common hepatic from the superior mesenteric, the guiding catheter has to be advanced through the aorta into the superior mesenteric artery (figure 7.04). The coaxial catheter is advanced into the common hepatic and then into the proper hepatic artery.

Figure 7.05a shows one variant in which the hepatic artery stems from the left gastric artery. This variant was reported from Michels' work but not observed in this study. For this vascular pattern, the endovascular procedure for SIR treatment has to be modified. In Michels' presented case, there was one more variant which warranted attention if the ^{90}Y-SIR treatment were to be carried out. The right gastric artery arising from the right hepatic artery. To avoid radiation induced gastritis, the right gastric artery has to be embolized first. Then infusion of the ^{90}Y microspheres could be proceeded on with the infusion site at the proper hepatic artery. According to the classification described in section 7.2, this is the 'Type II-a' endovascular treatment procedure. The procedures of embolizing the right gastric artery and positioning of the coaxial catheter tip at the appropriate infusion site in the hepatic artery is easier said than done. Both procedures involve manipulating the coaxial catheter a long distance. In the procedure for embolizing the right gastric artery, manipulation of the coaxial catheter into the right hepatic artery and into the right gastric artery to deliver the embolization coil requires expert skill. The theoretical placement of the catheters for ^{90}Y-SIR treatment is shown in figure 7.05b.

Figure 7.04 Position of the guiding catheter for 'CH fr SMA'

Trace drawing



From figure 6.10a

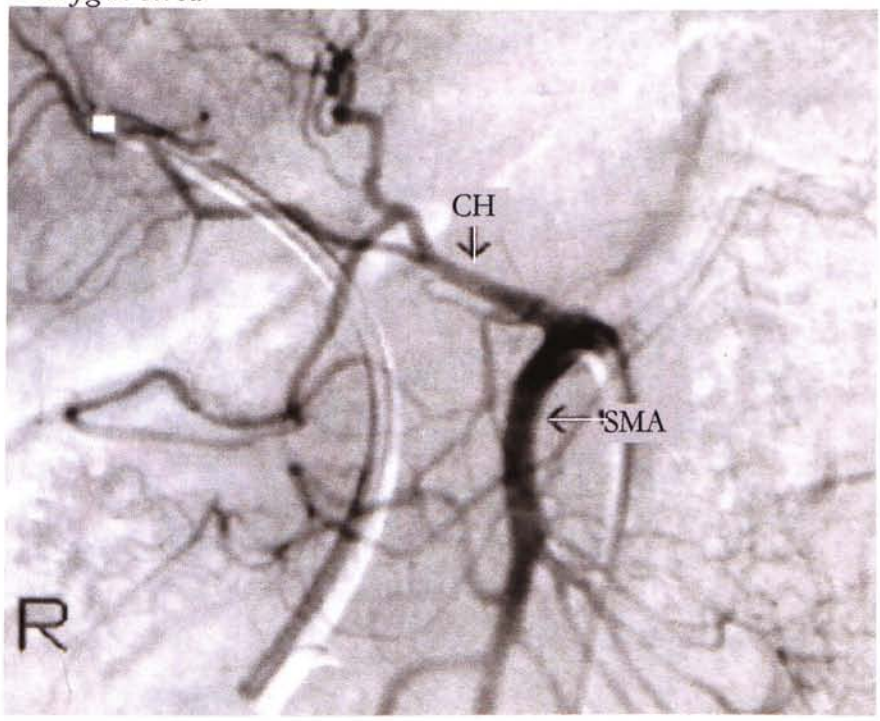


Figure 7.05a The hepatic artery takes origin from the left gastric artery
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)

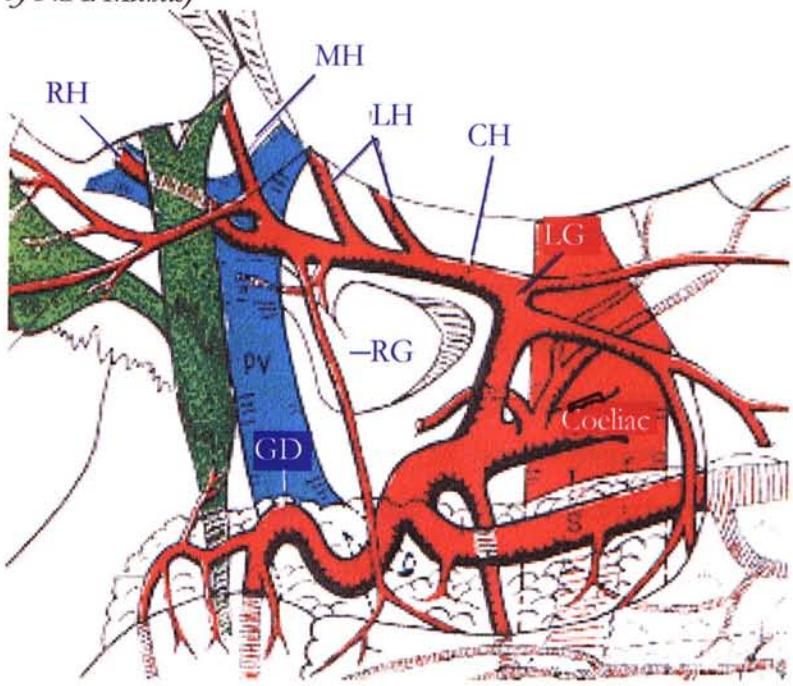
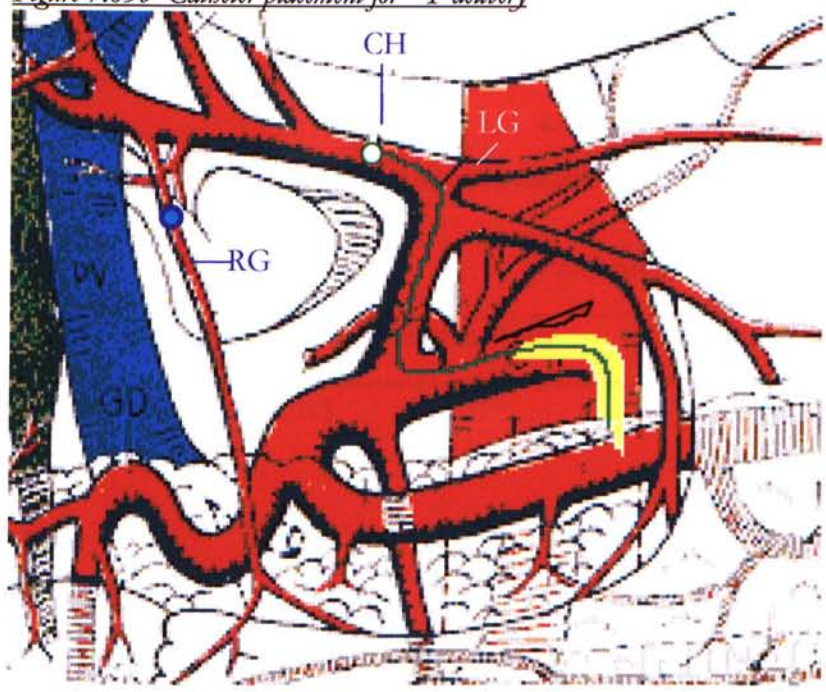


Figure 7.05b Catheter placement for ^{90}Y delivery



Guiding catheter
Coaxial catheter
Embolization coil
Infusion site

7.5 The right hepatic artery

In this study, the normal origin of the right hepatic artery is defined to be arising from the proper hepatic artery. All other sites of origin of the right hepatic artery or the existence of an accessory right hepatic artery are referred to as variants. In this study, 54 (19.6%) variant right hepatic arteries were encountered, while there were 52 (26%) variant right hepatic arteries encountered in Michels' study. Using the Chi-square test to compare the two, the p-value obtained is 0.12 which is greater than 0.05. This means that the two percentages do not differ significantly. Despite these apparently similar results, the percentages of the different variants do vary. Table 7.02 lists the percentages and the p-values of the different variants. Upon statistical testing, the percentages of a right hepatic artery arising from the superior mesenteric artery, a right hepatic artery arising from the aorta and an accessory right hepatic artery stemming from the superior mesenteric artery obtained in this study differ significantly from that reported in Michels' work. The three percentages in Michels' work are significantly greater than that in this study. These discrepancies may be due to racial difference but this topic is beyond the scope of this study.

Table 7.02 Comparison of the % of the variant origins of the right hepatic artery

	Percentage		
Vessel origin	**In this study**	**In Michels' work**	**p-value**
Superior mesenteric artery	4.7%	12.5%	0.003 <0.05
Coeliac	0.7%	3.0%	0.062
Aorta	0%	2.0%	0.031 <0.05
Left gastric	0%	0.5%	0.420
Accessory RH from SMA	1.1%	4.5%	0.041 <0.05

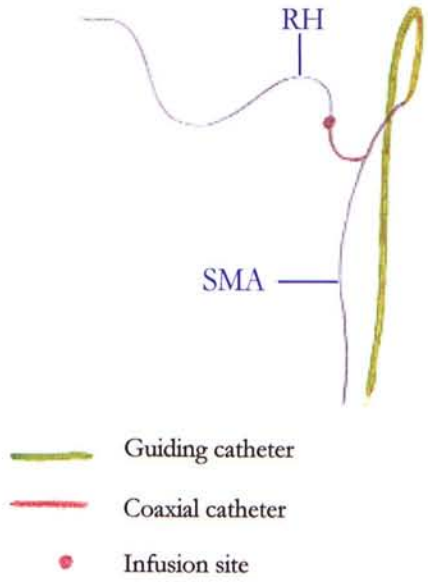
During the endovascular procedure for ^{90}Y-SIR treatment, for the 'normal' right hepatic artery arising from the proper hepatic, the catheter placement is as shown in figure 7.01. Modifications are needed in order that the ^{90}Y microspheres could be delivered to the entire arterial supply of the HCC when the right hepatic artery is displaced out of the main hepatic trunk or when an accessory right hepatic artery is present.

Figure 7.06 shows one example of a right hepatic artery originating from the superior mesenteric artery. The theoretical catheter placement is shown and the ^{90}Y microspheres is to be infused at the proximal end of the right hepatic artery. For this subject, at least one infusion of the ^{90}Y microspheres has to be given. The endovascular treatment procedure may be of 'Type I-a' or 'Type II' depending on the vascular anatomy of the other hepatic arteries and the related gastric vessels.

In the presence of an accessory right hepatic artery, theoretically, this accessory right hepatic has to be infused with ^{90}Y microspheres in addition to infusion at the main hepatic trunk. Figure 7.07 shows an example of an accessory right hepatic artery from the gastroduodenal. For this vascular pattern two infusions of the ^{90}Y microspheres are required, that is the 'Type I-b' endovascular treatment procedure can be applied. The catheter placement for infusing at the accessory right hepatic is demonstrated in the 'figure 7.07' and the other infusion site at the proper hepatic artery is illustrated in the same figure.

Figure 7.06 Catheter placement for ^{90}Y delivery for 'RH fr SMA'

Trace drawing



From figure 6.13a

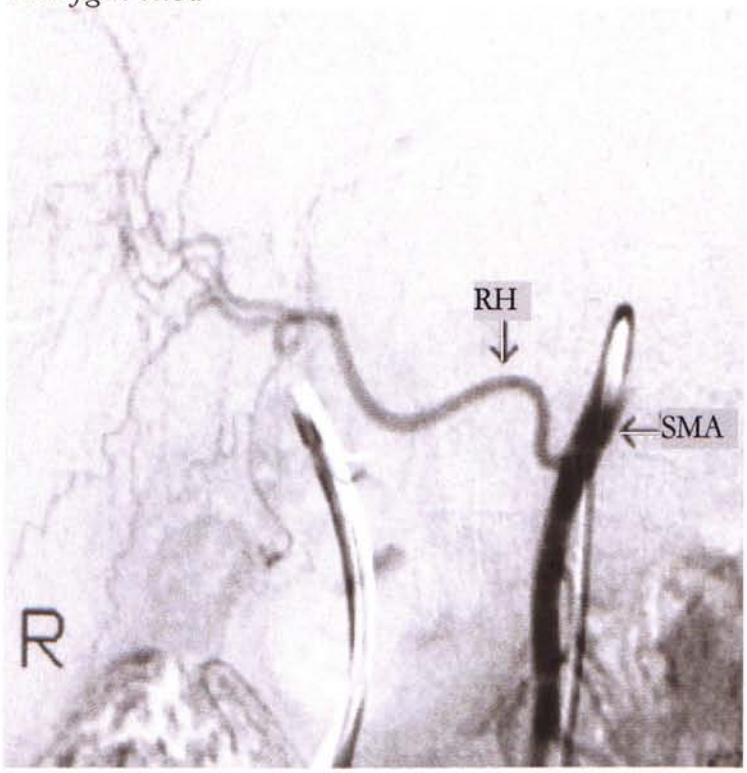
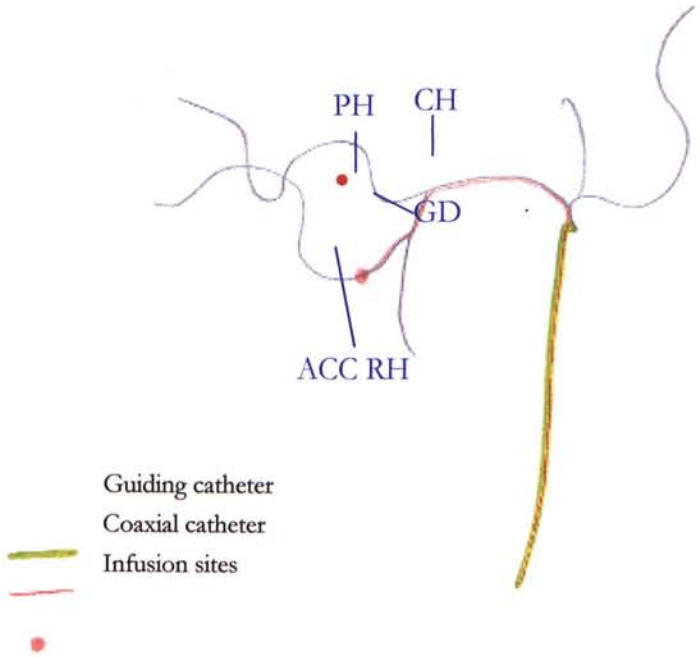
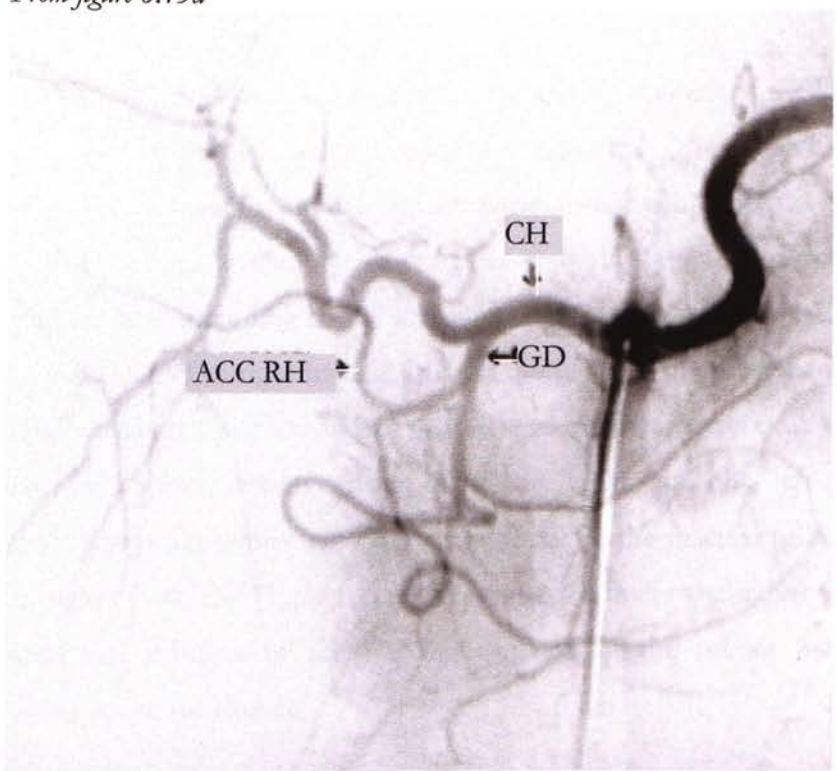


Figure 7.07 Catheter placement for ^{90}Y delivery for 'ACC RH fr GD'

Trace drawing



From figure 6.15a



7.6 The middle hepatic artery

According to anatomy textbook, the normal origin of the middle hepatic artery is the right hepatic or the left hepatic artery (figure 6.19a & 6.20a). In this study, 44.6% of the middle hepatic arteries arise from the right hepatic and 32.6% from the left hepatic artery. Corresponding figures quoted from Michels' work were both 45%. Upon statistical testing using Chi-square test, the p-values obtained are 0.94 and 0.59 respectively. This implies that the percentages obtained about the middle hepatic from the two studies do not differ significantly. In Michels' work, the remaining 6% of the middle hepatic arose from the coeliac, hepatic, gastroduodenal, or right gastric artery. Figure 7.08 is from Michels' text and according to his classification the origin of this middle hepatic is the right gastric artery. On the other hand, according to classification of this study, the middle hepatic artery is said to give rise to the right gastric artery instead of the other way round. Thus, direct comparison in this context of different classification cannot lead to any meaningful conclusion although the statistical value for comparison can be obtained.

The middle hepatic artery supplies the medial segment of the left lobe. Thus, the middle hepatic artery cannot be neglected when considering ^{90}Y-SIR treatment. When the middle hepatic artery arises from either the right or the left hepatic artery, any modification needed during the endovascular treatment procedure is according to the presentation of the right or the left hepatic artery respectively. The right hepatic artery is discussed in the previous section and the left hepatic is discussed in the next section. When there exist more than one middle hepatic arteries arising from the right and the left hepatic artery simultaneously, neither one can be neglected. In the vascular presentation shown in figure 7.09, the 'Type I-a' endovascular treatment procedure can be applied with one infusion of the ^{90}Y microspheres at the proper hepatic artery as illustrated in the sketch.

Other variant origins of the middle hepatic include the proper hepatic, the common hepatic and the gastroduodenal artery.

Figure 7.10 shows a middle hepatic artery arising from the proper hepatic. It is necessary to infuse the ^{90}Y microspheres at the proper hepatic artery prior to the branching off of the middle hepatic artery. For this subject, the left hepatic artery arises from the left gastric artery. If the tumour does not involve the left lobe of liver, the 'Type I-a' endovascular treatment procedure can be applied.

When considering the vascular presentation shown in figure 7.11 with the middle hepatic artery arising from the common hepatic artery, the 'Type II' endovascular treatment procedure is applied. Before the infusion of the ^{90}Y microspheres, the middle hepatic artery is embolized. Collaterals developed will carry the ^{90}Y microspheres to the medial segment of the left lobe and at the same time the ^{90}Y microspheres are prevented from going into the right gastric artery since this right gastric artery arises from the middle hepatic. This procedure is a combination of the 'Type II-a' and 'Type II-b' endovascular treatment procedure.

Another example of the combination of the 'Type II-a' and 'Type II-b' endovascular treatment procedure is shown in figure 7.12. Here, the middle hepatic artery arises from the gastroduodenal. Before infusion of the ^{90}Y microspheres at the right hepatic artery, the gastroduodenal artery is embolized at the proximal end. The collaterals developed will infuse the medial segment of the left lobe of the liver and the gastroduodenal artery is protected. If the tumour also involves the left lobe, the tiny left hepatic has to be embolized prior to treatment.

Figure 7.08 The middle hepatic stemming from the right gastric artery
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)

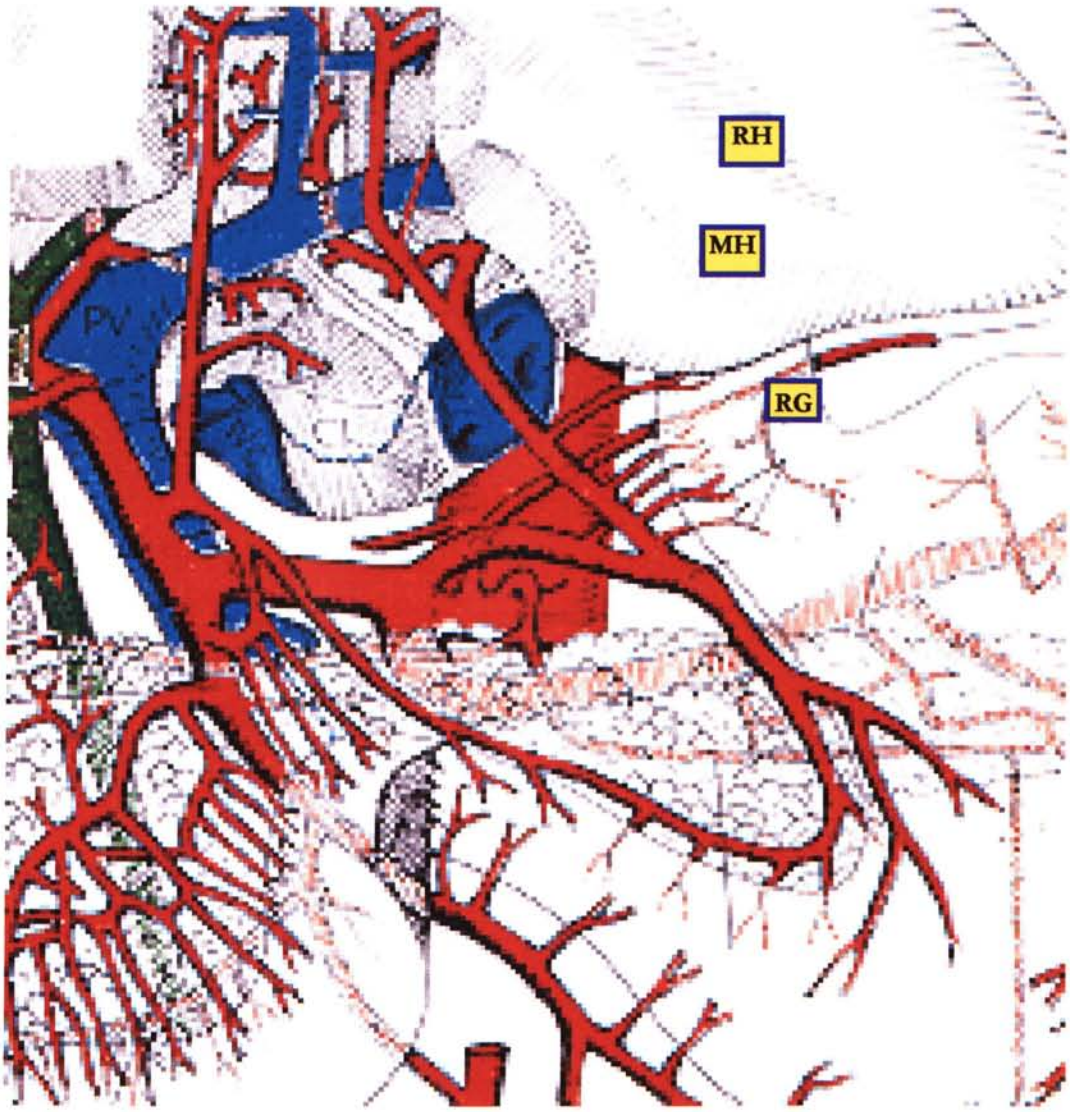
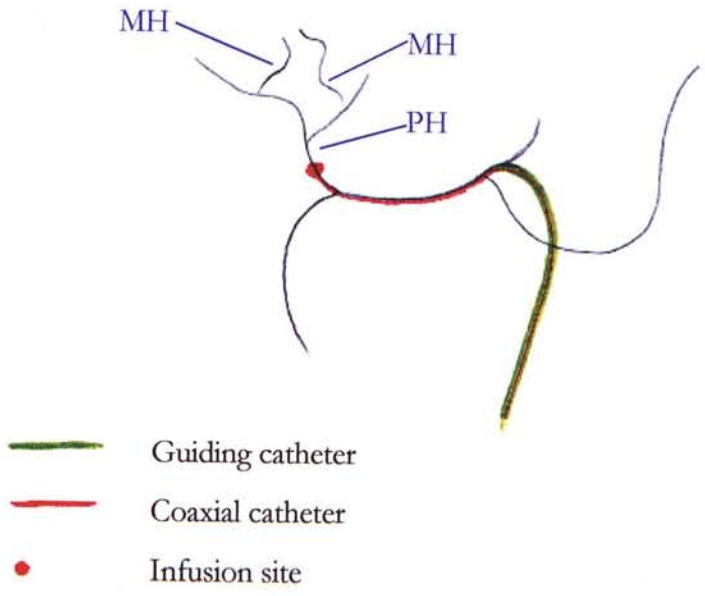


Figure 7.09 Catheter placement for ^{90}Y delivery for 'MH fr RH & LH'

Trace drawing



From figure 6.21a

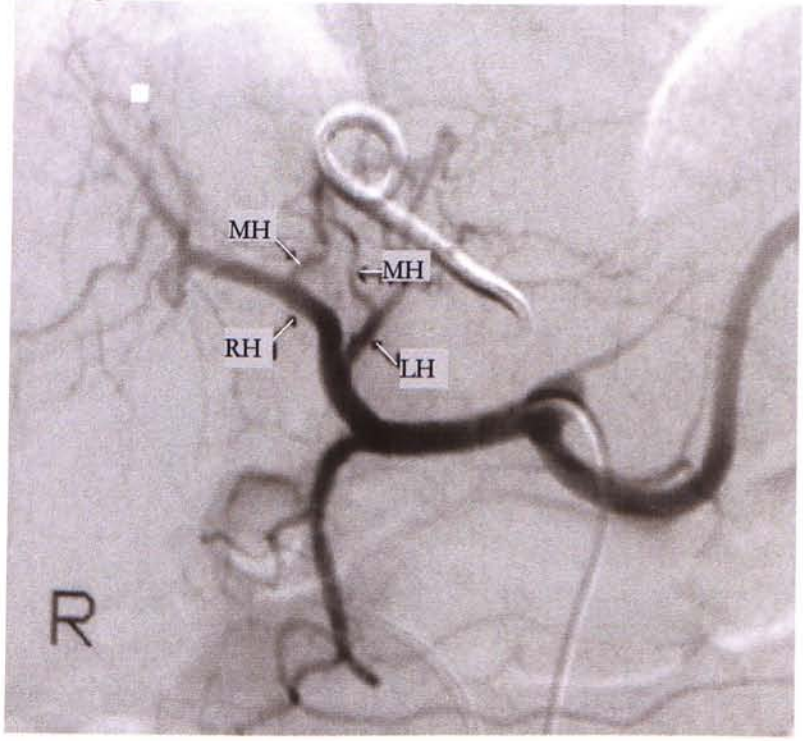
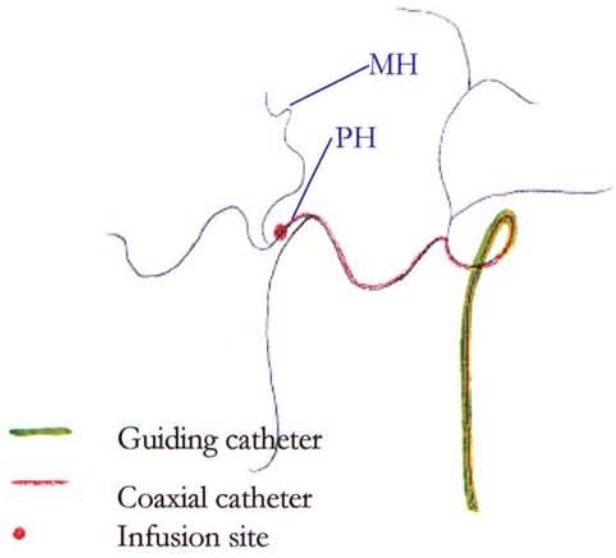


Figure 7.10 Catheter placement for ^{90}Y delivery for 'MH fr PH'

Trace drawing



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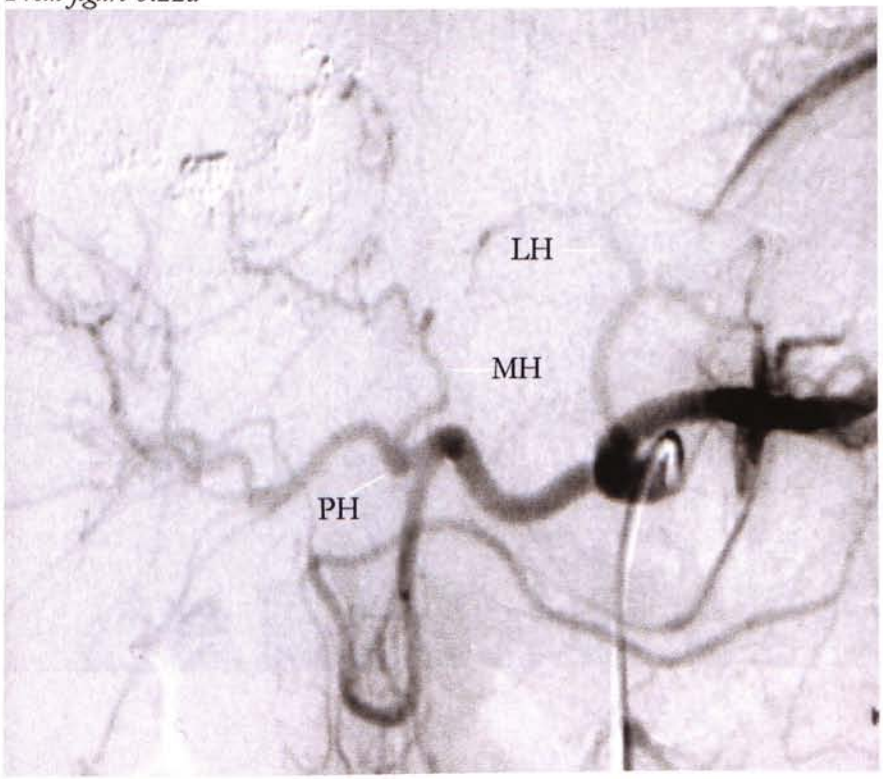
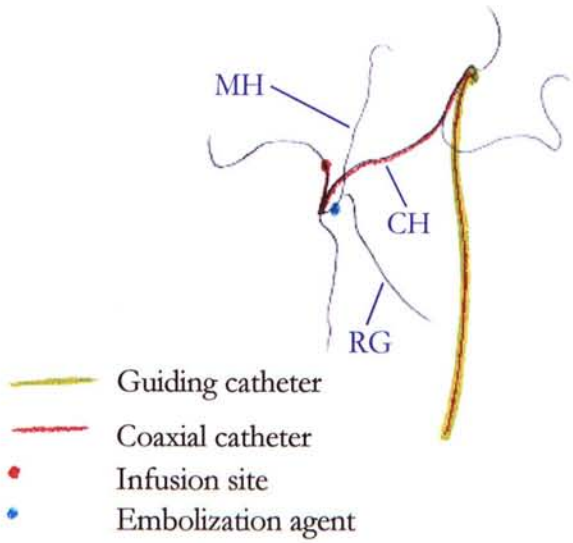


Figure 7.11 Catheter placement for ^{90}Y delivery for 'MH fr CH'

Trace drawing



From figure 6.23a

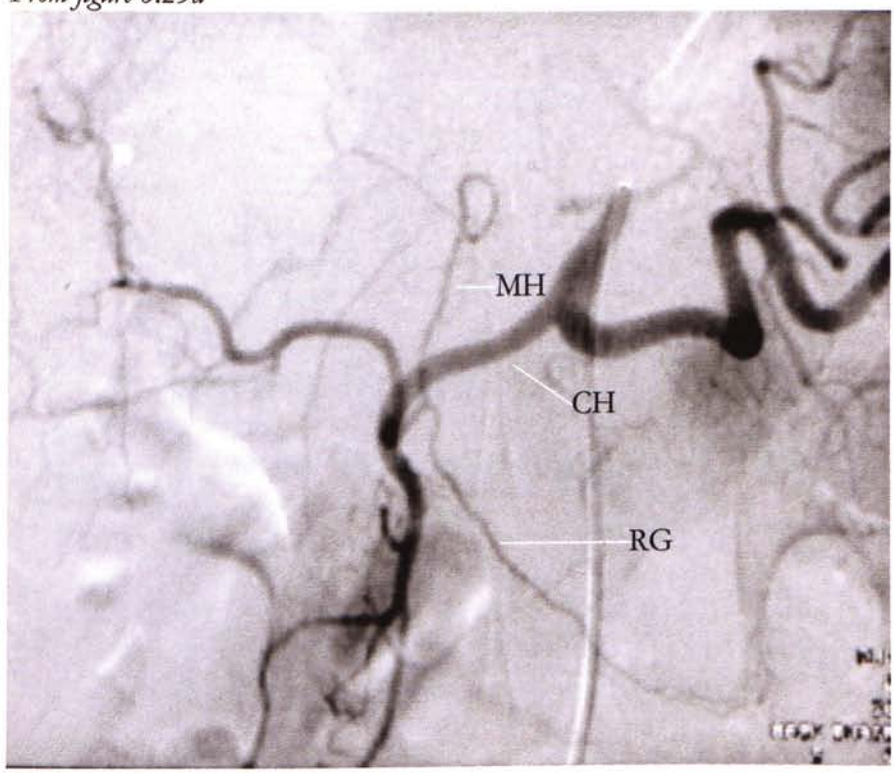
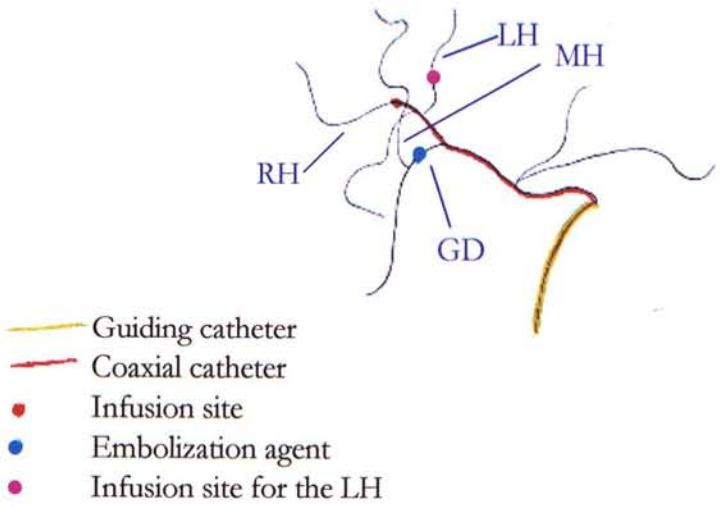


Figure 7.12 Catheter placement for ^{90}Y delivery for 'MH fr GD'

Trace drawing



From figure 6.24a

7.7 The left hepatic artery

As previously stated, the normal origin of the left hepatic is the proper hepatic. In this study, 77.5% of the subjects have this normal origin. Other variant origins include common hepatic artery, left gastric artery, and coeliac axis. Sometimes the left hepatic artery is absent or an accessory left hepatic artery may arise from the left gastric artery. When comparing with Michels' work, only two pairs of percentages are available and they are tested using the Chi-square statistical test. The p-values are listed in the following table (Table 7.03).

Table 7.03 Comparison of the % of the variant origins of the left hepatic artery

	Percentage		
Vessel origin	**In this study**	**In Michels' work**	**p-value**
Left gastric artery	7.2%	11.5%	0.15
Accessory LH from left gastric	4.0%	11.5%	0.003 <0.05

One significant difference observed is that the percentage of an accessory left hepatic artery arising from the left gastric in Michels' work is much higher than that in this study.

In 1.5% of the subjects in Michels' study, the left hepatic arose from the aorta. None was observed in this study. In this study, no extra radiation exposure was imparted to the individuals, each individual had a coeliac arteriogram and a superior mesenteric arteriogram routinely and the arteriograms were analysed retrospectively. There was one subject with absent left hepatic artery. This meant that the individual did not have a left hepatic artery from the coeliac axis or from the superior mesenteric artery. The left hepatic artery might take origin from the aorta which was missed in this particular case. Thus, direct comparison of the

results from these two studies did not lead to meaningful implication when the research methods employed were different.

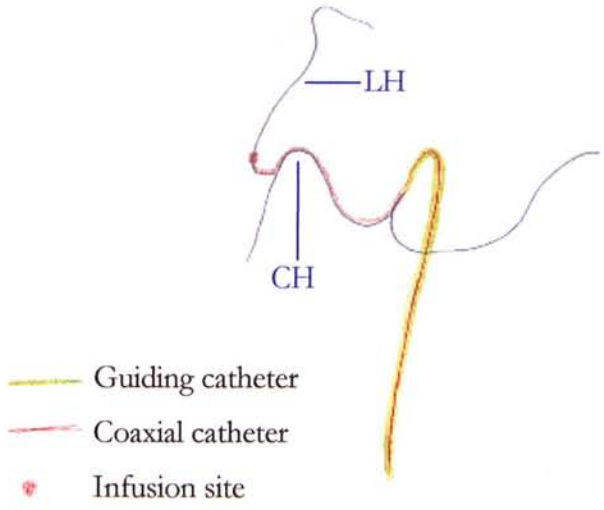
Variant left hepatic can be grouped into two types, the left hepatic being displaced from the coeliac axis and the presence of an accessory left hepatic. Generally speaking, the 'Type I-b' or the 'Type II' endovascular treatment procedure is applicable depending on the origin of the left hepatic artery or the accessory left hepatic artery and the presentation of the other hepatic arteries.

Figure 7.13 and figure 7.14 show a left hepatic artery from the common hepatic artery and from the left gastric artery respectively. Unless the left lobe of the liver is spared from the tumour, the left hepatic artery has to be infused with ^{90}Y microspheres at its proximal end in addition to infusion for the other hepatic arteries.

The existence of an accessory left hepatic amounts to 4%. As a basic principle, infusion is preferred to embolization since active treatment is preferred to passive treatment. That is 'Type I-b' treatment procedure is preferred to 'Type II-b' treatment procedure. The ground rule is to infuse the accessory left hepatic separately during the endovascular treatment procedure unless the accessory left hepatic is small in calibre. Figure 7.15 shows an accessory left hepatic arising from left gastric artery. The calibre of the accessory left hepatic is relatively smaller than that of the left gastric. Infusion of radioactive drug into the small accessory left hepatic might lead to reflux into the left gastric which in turn will cause radiation gastritis. Thus, it is wise to embolize the accessory left hepatic allowing the collaterals to infuse the territory supplied by the accessory left, and to infuse the ^{90}Y microspheres at the proper hepatic artery.

Figure 7.13 Catheter placement for ^{90}Y delivery for 'LH fr CH'

Trace drawing



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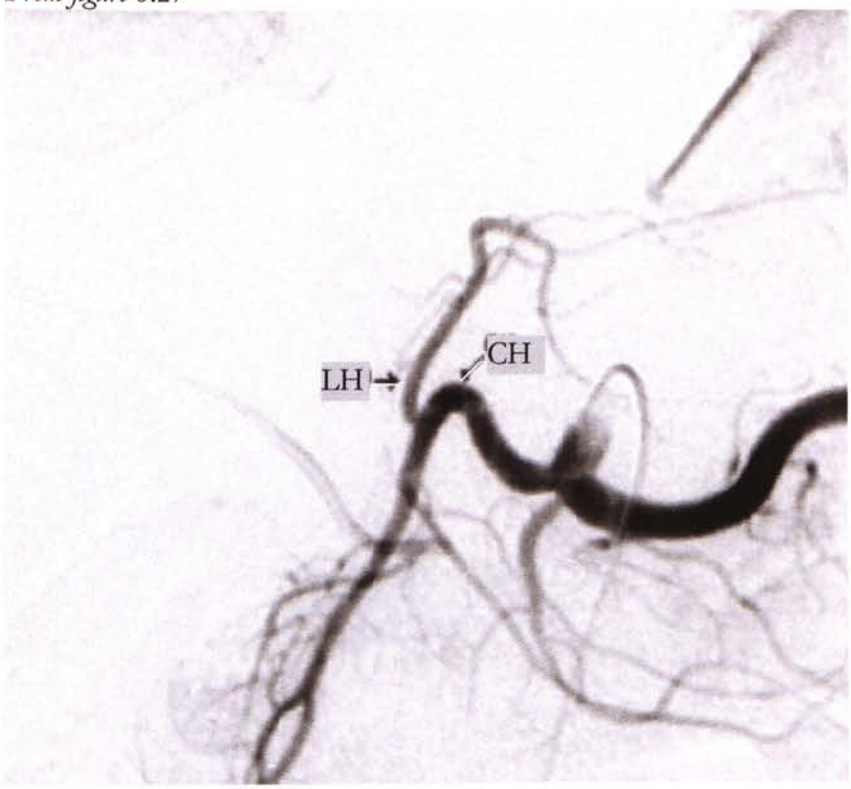
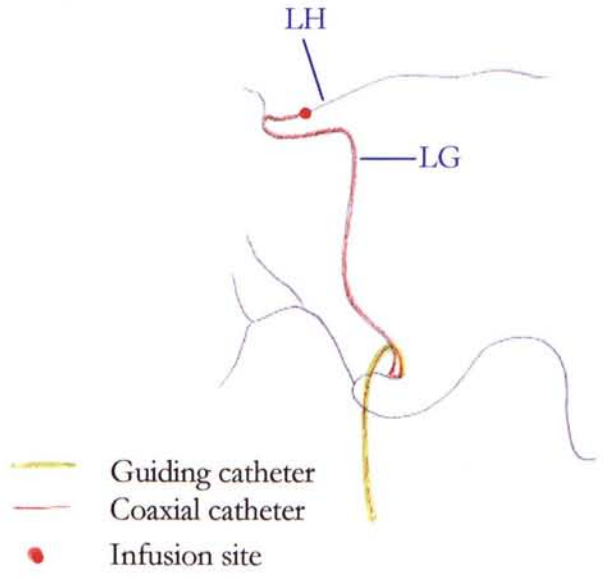


Figure 7.14 Catheter placement for ^{90}Y delivery for 'LH fr LG'

Trace drawing



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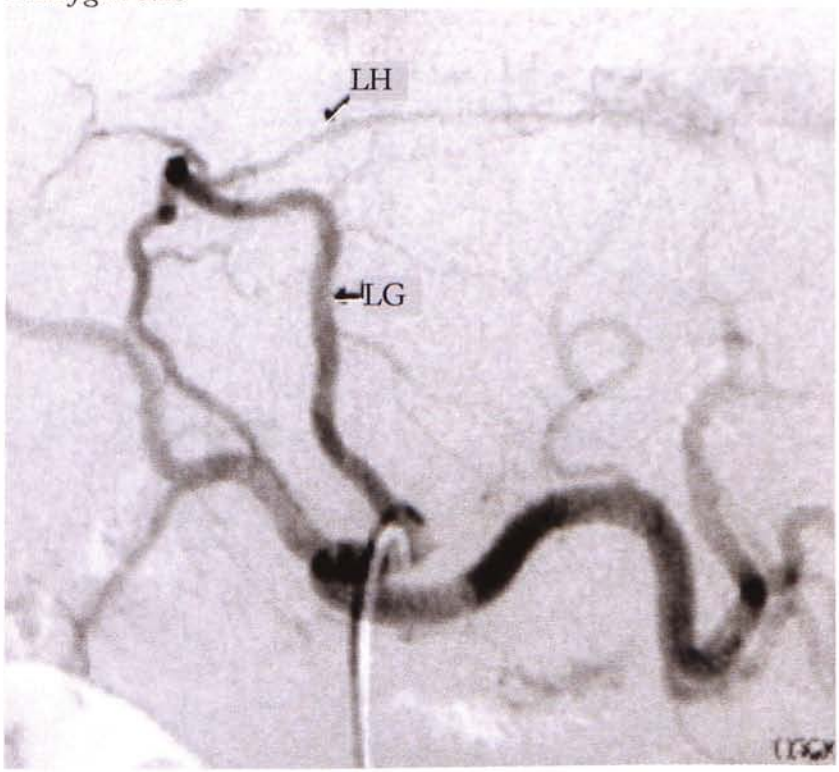
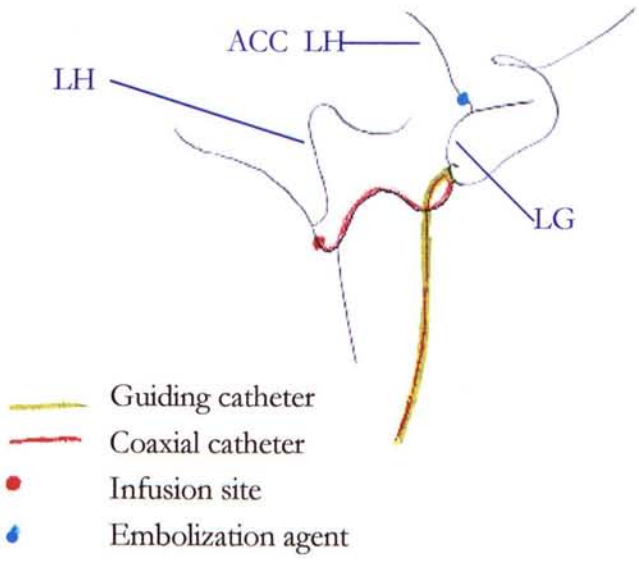
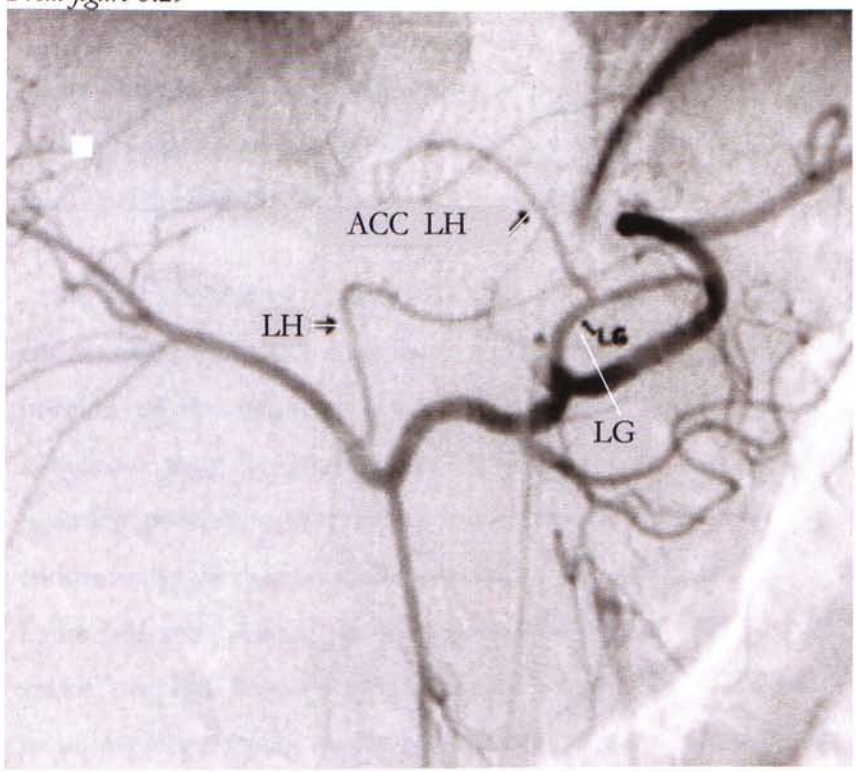


Figure 7.15 Catheter placement for ^{90}Y delivery for 'ACC LH fr LG'

Trace drawing



From figure 6.29



7.8 The Gastroduodenal artery

The anatomy text book normal origin of the gastroduodenal artery is the common hepatic artery. In this study, 93.1% of the individuals presented with this origin. Other variant origins include right hepatic, left hepatic and coeliac axis. The following table (Table 7.04) lists the p-values and the different percentages about the gastroduodenal artery obtained from this study and Michels' study.

Table 7.04 Comparison of the % of the variant origins of the gastorduodenal artery

	Percentage		
Vessel origin	**In this study**	**In Michels' work**	**p-value**
Right hepatic artery	5.1%	7.0%	0.49
Left hepatic artery	1.4%	11.0%	0.00001 < 0.05
Coeliac axis	0.4%	1.5%	0.20

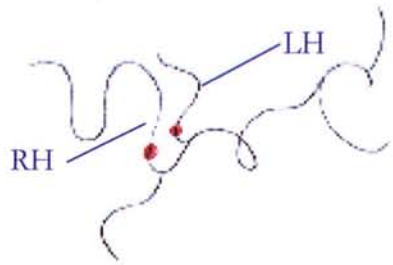
In Michels' work, the percentage of a gastroduodenal arising from the left hepatic is significantly higher than that obtained in this study. The explanation of this big difference is beyond the scope of this study.

In the context of this study, the way the variant vessels influence the endovascular treatment procedure is the main concern. To know the exact position of the origin of the gastroduodenal artery is to avoid introducing radioactive drug to the stomach via the gastroduodenal artery during ^{90}Y-SIR treatment procedure. For the 'normal' origin of the gastroduodenal, the 'Type I-a' endovascular procedure can be applied. The catheter placement is shown in figure 7.01 with infusion site at the proper hepatic provided that the entire hepatic arterial supply is from the proper hepatic artery. Otherwise, modifications have to be made according to the presentation of the hepatic arteries and the right gastric artery.

When the gastroduodenal artery arises from the right or the left hepatic artery, generally the 'Type I-b' endovascular treatment procedure can be applied. At least two infusions of the ^{90}Y microspheres, one at the right hepatic and the other at the left hepatic, are to be given and the infusion sites are to be distal to the branching off of the gastroduodenal from either the right or the left hepatic artery. Figure 7.15, figure 7.16, figures 7.17a & 7.17b demonstrate the theoretically best infusion sites.

Figure 7.16 Catheter placement for ^{90}Y delivery for 'GD fr RH'

Trace drawing



Infusion site

From figure 6.32

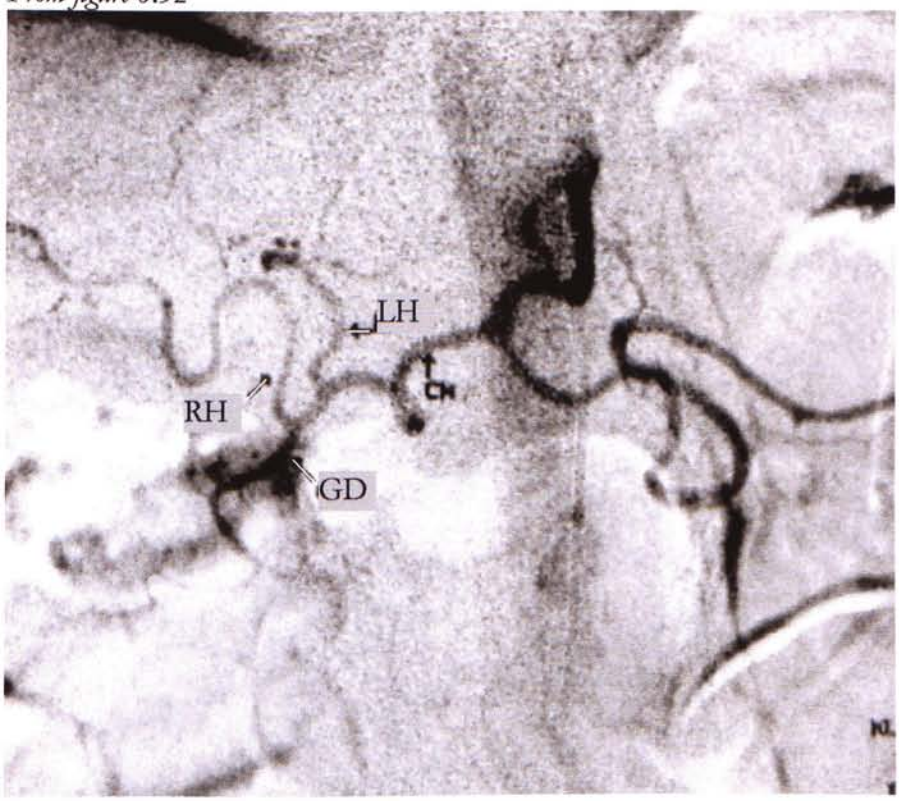
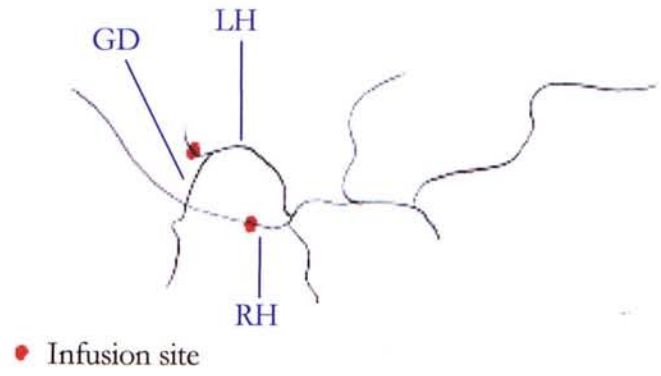


Figure 7.17a Catheter placement for ^{90}Y delivery for 'GD fr LH'

Trace drawing



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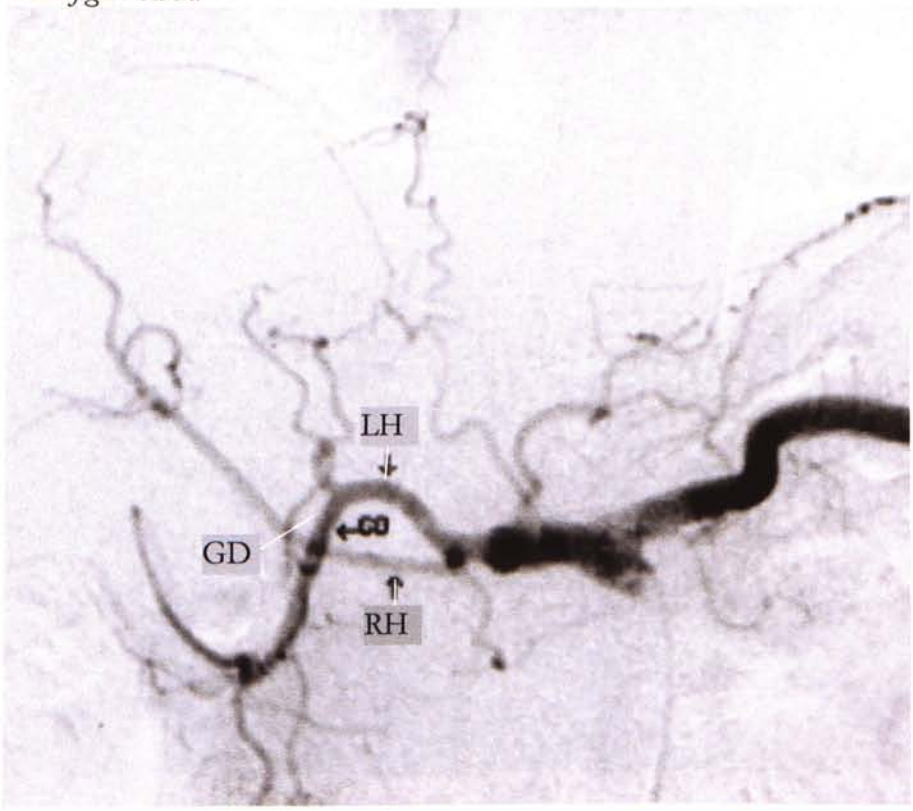
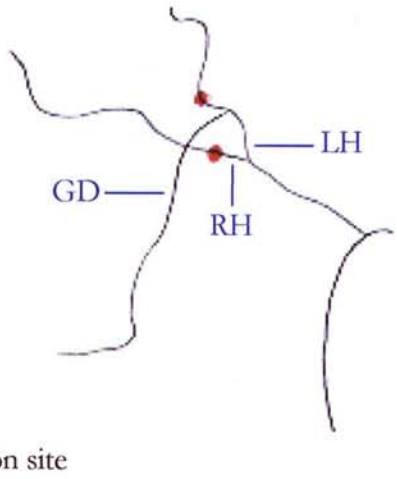
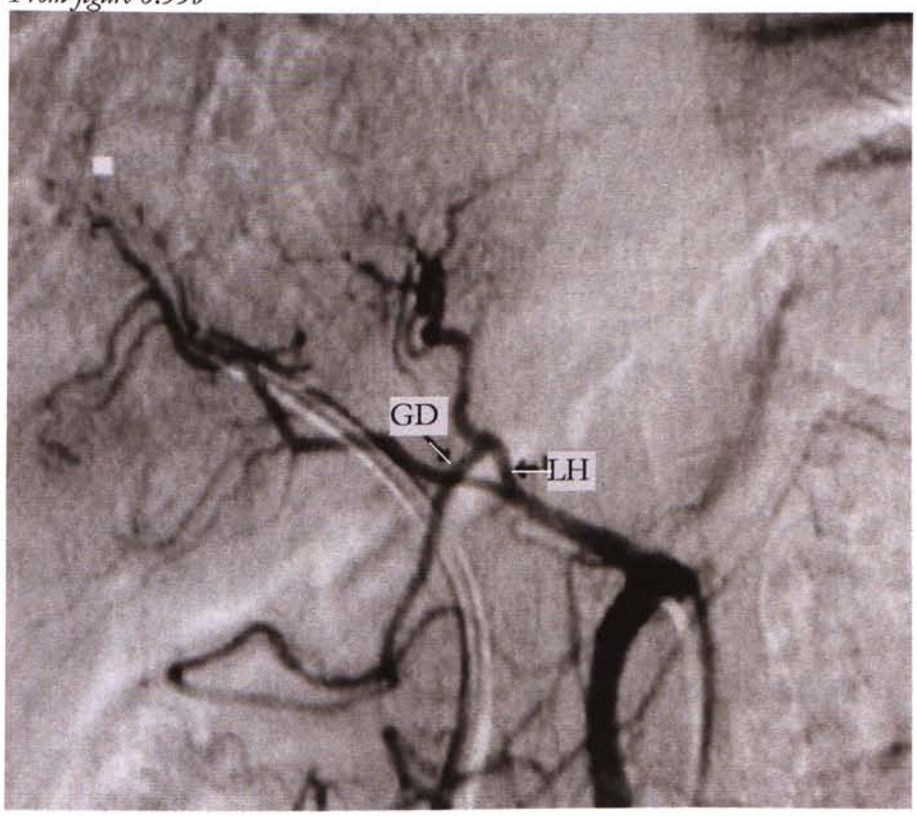


Figure 7.17b Catheter placement for ^{90}Y delivery for 'GD fr LH'

Trace drawing



From figure 6.33b



7.9 The right gastric artery

The varied mode of the origin of the right gastric is due to the varied mode in which the hepatic artery gives off its main branches. Most commonly, the right gastric arises from the proper hepatic artery. (Michels, 1955) In this study, 42% of the individuals have the right gastric artery originating from the proper hepatic artery. Obviously, all other variants amount to 58% which means that more than half of the individuals have variant right gastric artery. This seems to be the artery with the most variable origin and as the capillary bed is of great concern this artery will be the most likely to cause anxiety during SIR. Due to different method of classification, the available percentages for comparison are listed in table 7.05.

Table 7.05 Comparison of the % of the variant origins of the right gastric artery

	Percentage		
Vessel origin	**In this study**	**In Michels' work**	**p-value**
Left hepatic artery	35.5%	40.5%	0.30
Right hepatic artery	10.1%	5.5%	0.09
Gastroduodenal artery	3.6%	8.0%	0.06
Absent	2.2%	0%	0.03 < 0.05
Middle hepatic artery	1.1%	5.0%	0.02 < 0.05

A significant difference was observed in the percentage of a right gastric artery arising from the middle hepatic artery. The right gastric artery is small and characteristically filamentous with an average diameter of 2mm. The 2.2% absent right gastric artery in this study may be due to the small size of the right gastric artery.

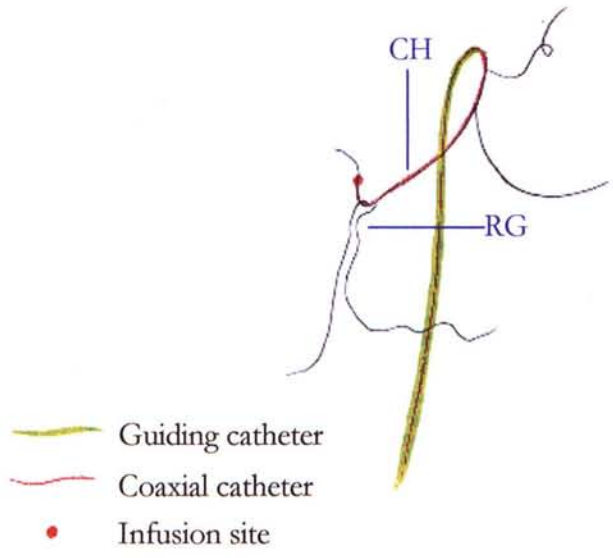
In this context, the variant right gastric artery can be divided into two groups. All variant origins which are proximal to the normal origin form one group. The other variant origins which are distal to the normal origin form another group.

Figure 7.18, figure 7.19, and figure 7.20 are examples of the former group of variant right gastric artery. The type of endovascular treatment procedure is predominantly governed by the vascular presentation of the hepatic arteries. The variant right gastric in this group has no significant influence in this respect.

For the latter group where the variant origin of the right gastric artery is distal to the normal origin, the influence on the endovascular procedure is described here. Usually, the 'Type I-b' or the 'Type II-b' endovascular treatment procedure can be applied when ^{90}Y-SIR treatment has to be given. Figure 7.21 shows a right gastric from the left hepatic and the 'Type I-b' endovascular treatment method is applied with one infusion of radioactivity at the right hepatic and the other infusion at the left hepatic. Figure 7.22 shows a right gastric from the right hepatic and the 'Type II-b' method is applied with embolization of the right gastric artery preceded by infusion of the radioactivity at the proper hepatic. Figure 7.23 shows a right gastric from the bifurcation of the proper hepatic and the 'Type I-b' method is most suitable with two infusions of the radioactivity at the right and the left hepatic arteries.

Figure 7.18 Catheter placement for ^{90}Y delivery for 'RG fr CH'

Trace drawing



From figrue 6.37a

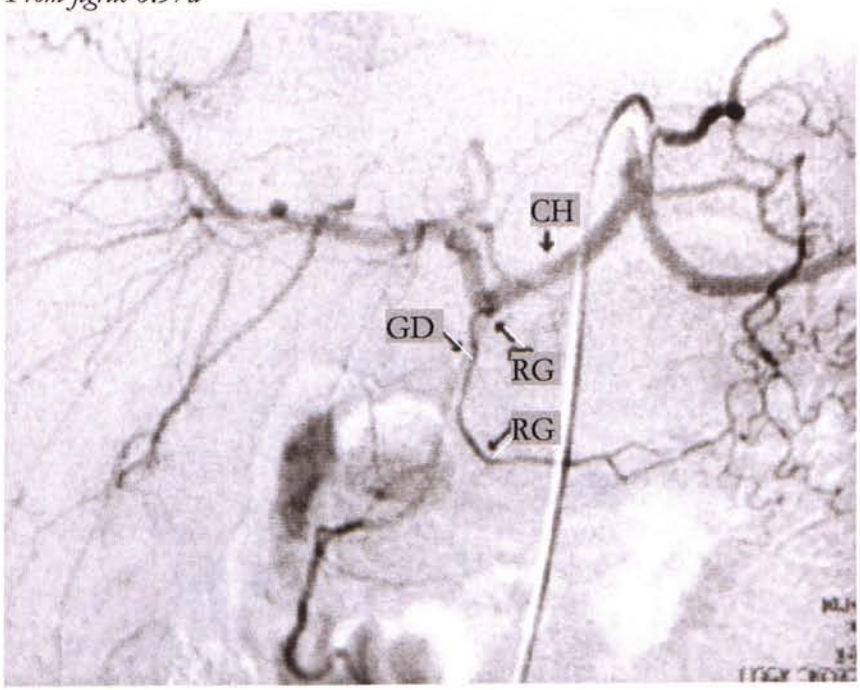
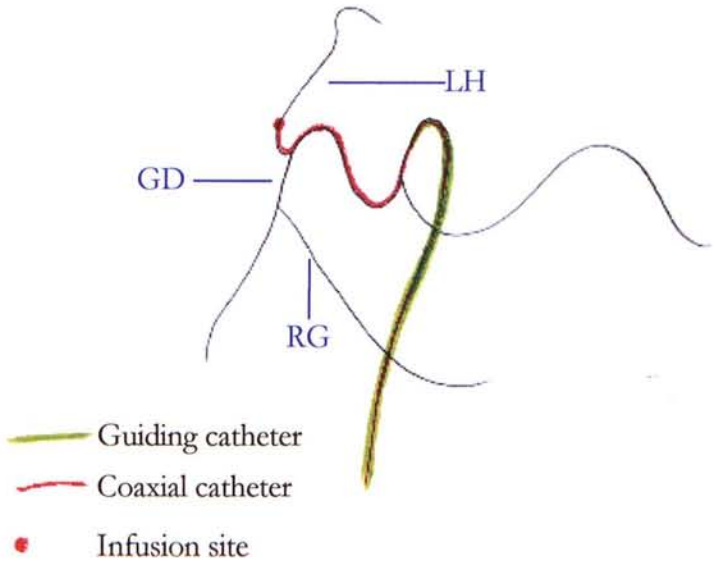


Figure 7.19 Catheter placement for ^{90}Y delivery for 'RG fr GD'

Trace drawing



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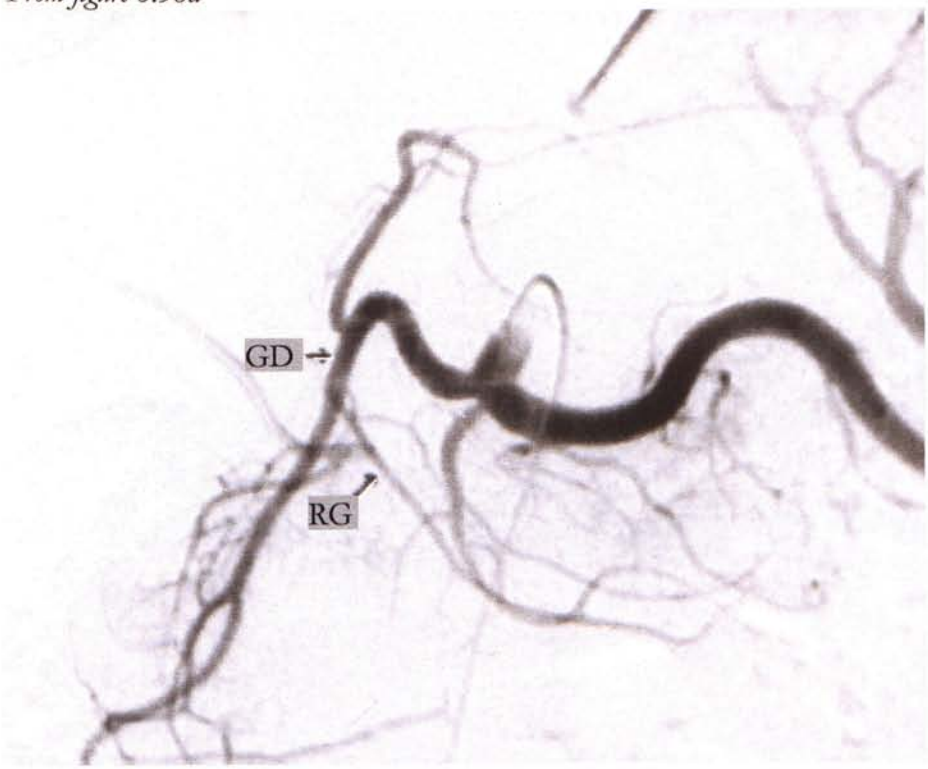
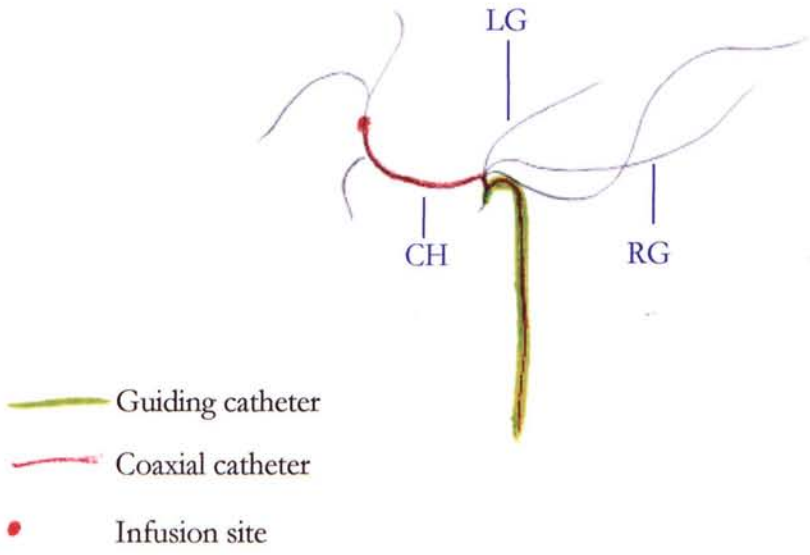


Figure 7.20 Catheter placement for ^{90}Y delivery for 'RG fr Bif of CH'

Trace drawing



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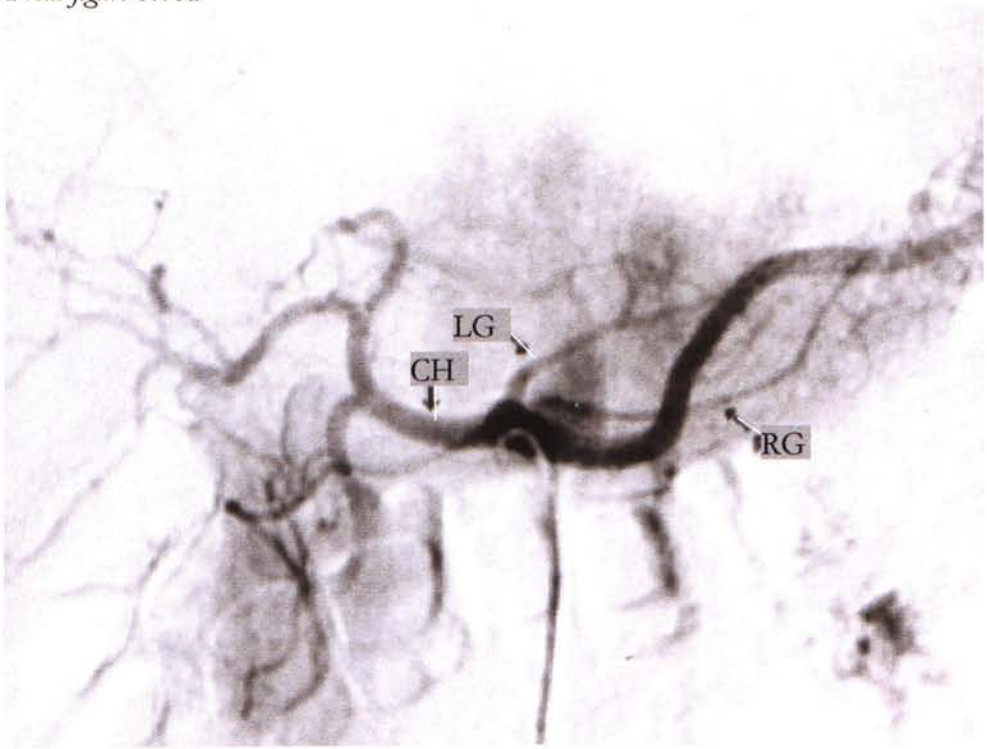
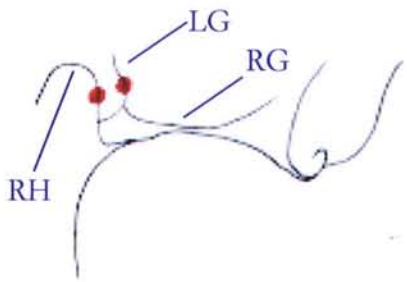


Figure 7.21 Catheter placement for ^{90}Y delivery for 'RG fr LH'

Trace drawing



● Infusion site

From figure 6.35a

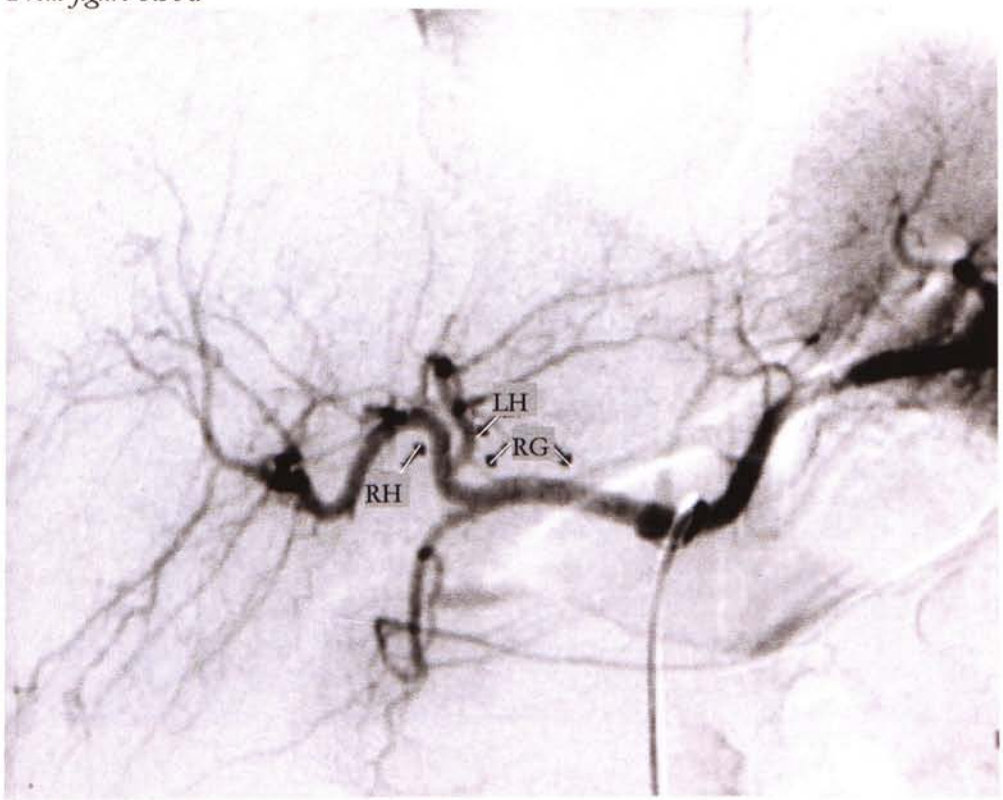
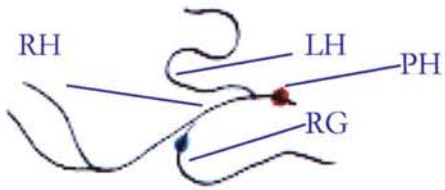


Figure 7.22 Catheter placement for ^{90}Y delivery for 'RG fr RH'

Trace drawing



- Infusion site
- Embolization agent

From figure 6.36a

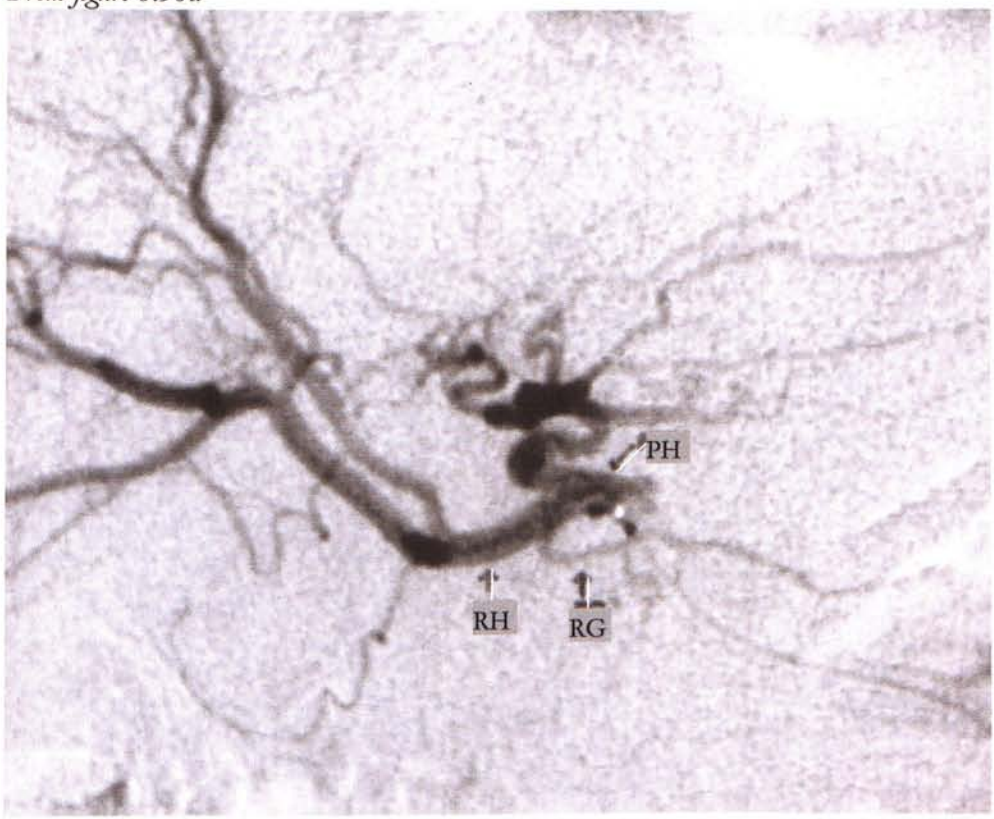
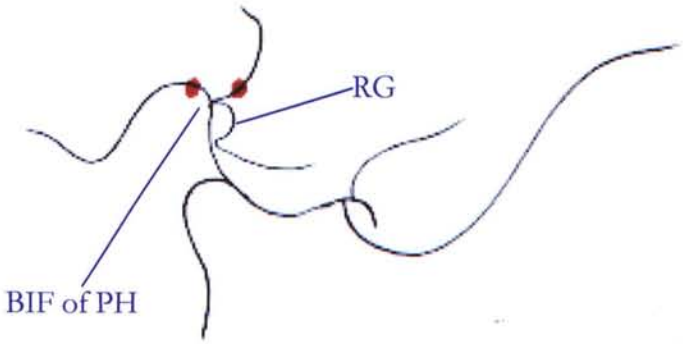


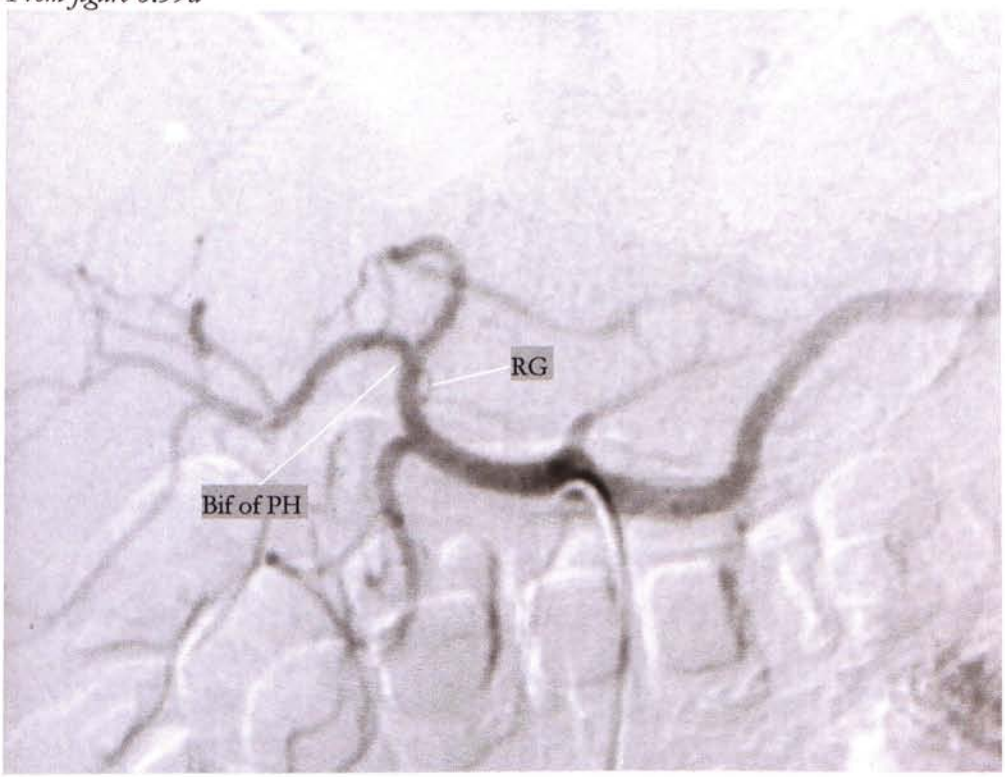
Figure 7.23 Catheter placement for ^{90}Y delivery for 'RG fr BIF of PH'

Trace drawing



● Infusion site

From figure 6.39a

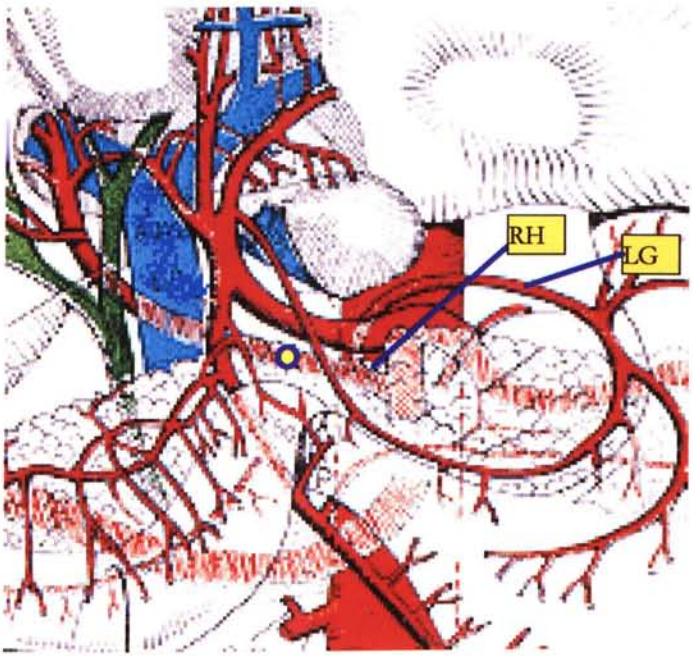


7.10 The left gastric artery

Classically the left gastric artery is the first branch of the coeliac axis, thus it is included in this study. The variant origins of the left gastric artery observed in this study include aorta and bifurcation of the coeliac axis. Sometimes the left gastric artery is absent. None of these variant origins have special influence on the ^{90}Y-SIR treatment procedure as long as the left gastric origin is proximal to the infusion site.

One odd pattern was observed in Michels' study where the left gastric artery took origin from a right hepatic artery (figure 7.24). If endovascular treatment procedure were prescribed, modification had to be in such a way that the infusion should be distal to the origin of the anomalous left gastric artery.

Figure 7.24 Left gastric originating from the right hepatic artery
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)



Infusion site at the RH

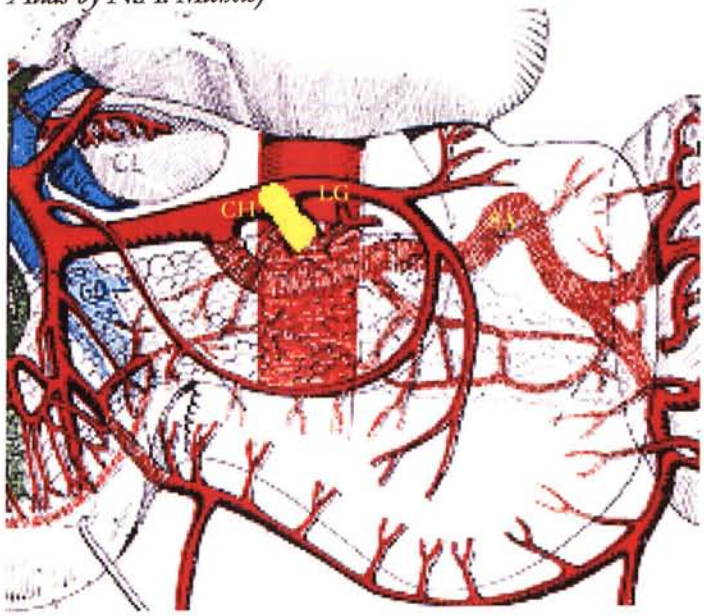
7.11 The splenic artery

The splenic artery stems from the coeliac axis according to anatomy text books. In this study 99.3% of the subjects have this normal presentation. Michels quoted that 82% splenic artery arose from the coeliac via a hepatolienogastric trunk. This apparently very different result may be due to racial difference, difference in research method, difference in classification method or others. Figure 7.25a and figure 7.25b demonstrate one example of different classification. Michels classified the splenic artery shown in figure 7.25a as a branch from the common hepatic. While in this study, this splenic is defined to be stemming from the coeliac axis as illustrated in figure 7.25b. The difference of the two studies is not the main theme of this study, but is a good illustration of the difficulties of classification, particularly where a trifurcation is involved.

Other variant origins of the splenic artery observed in the study are the aorta and the superior mesenteric artery. These variants do not have any significant influence on the endovascular treatment procedure of ^{90}Y-SIR. .

Figure 7.25a Splenic artery originating from the common hepatic artery

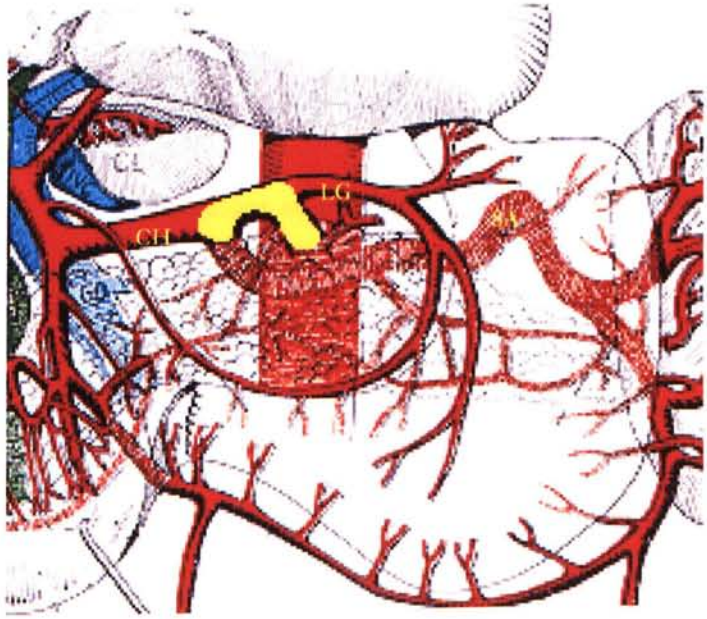
(From Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)



Coeliac axis

Figure 7.25b Splenic artery originating from the coeliac axis

(Modified from Blood Supply and Anatomy of the Upper Abdominal Organs With a Descriptive Atlas by N.A. Michels)



Coeliac axis

7.12 Comparison with the golden classics

Percentages observed in this study and percentages quoted from Michels were compared. There were only 25 pairs of values for comparison. The Chi-square test was used to test 13 pairs of the values and the other 12 pairs was tested with the Fishers' Exact Test since the expected cell frequency of these 12 pairs were ≤ 5. The p-values obtained were listed in Appendix VIII. There were seven p-values which were less than 0.05. The corresponding seven pairs of percentages differed significantly and all the seven percentages from Michels' work were greater than that observed in this study. These seven variants were, right hepatic from superior mesenteric, accessory right hepatic from superior mesenteric, right hepatic from aorta, accessory left hepatic from left gastric, left hepatic from superior mesenteric, gastroduodenal from left hepatic, and right gastric from middle hepatic. The comparisons of these seven variants were shown in table 7.06.

Michels speculated a few causative factors for arterial variations, viz :

- Variations in constitutional inheritance of which the anatomists know practically nothing for the body of the father or the mother of the body studied is never dissected.

- Variations of evolution, i.e. atavistic remnants, of either a progressive or a regressive nature. This is impossible route for the human species because of lack of material.

- Variations in hemo-dynamic potential, the type and the intensity of activity of a given organ and its rate of growth determining the constitution and the distribution of the arterial pattern (Thoma, Mall).

- Variations of racial differences.

- Ontogenetic developmental peculiarities. The patterns of the arteries are determined by internal and external factors. For the coeliac axis and the superior mesenteric artery, developmental peculiarities would be correlated with the followings:

  1. variations in the degree and the site of gut rotation,

  2. persistence of differently interrupted sections of the primitive roots of the omphalomesenteric arteries and their longitudinal anastomoses (Tandler, 1904),

  3. variations in the rate and the manner of the cephalocaudad migration of the primitive gut arteries, as first claimed by Mall in 1891.

Of the speculated causative factors, racial difference might explain the difference observed in the two studies. Despite the fact that the percentages quoted from Michels are greater than that obtained in this study, the conclusion that 'arterial variations of the coeliac axis is more extensive in European than in Hong Kong Chinese' cannot be made since differences in the classification method, the method of research, the objectives of the research and the point of view of the researchers are there to invalidate this conclusion. The main difference of the two researches are tabulated in table 7.07.

However regardless the arteries in which there is no disagreement in classification, and technical aspects of visualization or demonstration of the artery are unlikely to lead to significant misses there does seem to be sufficient grounds to state that there is a racial difference. This is not only of interest from a purely anthropological point of view but as has been amply demonstrated, is of great medical importance.

Table 7.06 P-values of the seven 'different' variants, as compared with Michels

Vessel	Vessel origin	% from this study	% from Michels	p-value
RH	SMA	4.7%	12.5%	0.003 < 0.05
ACC RH	SMA	1.1%	4.5%	0.042 < 0.05
RH	Ao	0%	2.0%	0.031 < 0.05
ACC LH	LG	4.0%	11.5%	0.003 < 0.05
LH	SMA	0%	2.5%	0.013 < 0.05
GD	LH	1.4%	11.0%	0.00001 < 0.05
RG	MH	1.1%	5.0%	0.021 < 0.05

Table 7.07 Difference of the two researches, Michels and this study

		Michels' study (Michels, 1955)	This study
Classification method	:	Variants were grouped into types and counts obtained for statistical study	Counts of individual variant origin of vessels were obtained
Research method	:	Vascular patterns were obtained from dissection of cadavers	Vascular patterns were obtained from digital subtracted arteriograms
Research objectives	:	To provide a comprehensive atlas of vascular variants for surgeons	To establish percentages of individual variant origin and to find the influence of the variants on the SIR procedure
Point of view	:	From the point of view of an anatomist	From the point of view of a radiological interventionist

7.13 Comparison of subjects with HCC and without HCC

In the study, there were 166 subjects with HCC and 110 subjects with other upper abdominal lesions without HCC. The Chi-square and Fishers Exact Tests were used to test the two groups for any statistical significance. All the p-values obtained are greater than 0.05 and all the p-values are listed in Appendix XIV. This implies that the group with HCC and the group without HCC do not differ significantly in arterial variations of the coeliac axis and its branches. Therefore the HCC group were not a selected sample with regards the study of anatomy and anatomical variants and conversely that there was no predilection to developing HCC as a result of particular anatomical arterial pathways.

7.14 Comparison of the male group and the female group

Among the subjects recruited in the study, 197 were males and 79 were females. Chi-square and Fishers' Exact tests were used to test the two groups. All the p-values (see Appendix VX) are greater than 0.05 which means that statistically the two groups do not differ significantly. Thus, we can conclude that there is no gender difference in arterial variations of the coeliac axis and its branches.

Chapter 8

CONCLUSIONS

Trans-catheter ^{90}Y-SIR therapy is a feasible treatment for inoperable HCC. (W.Y.Lau *et al.*, in press). This ^{90}Y-SIR therapy sheds new light on the difficult HCC and at same time brings the attention of the clinical oncologist and the radiological interventionist to the vascular anatomy of individual patient who is otherwise unsuitable for the treatment procedure. In this study, the vascular anatomy of the hepatic arterial supply is scrutinized. 72% of the population of the Hong Kong Chinese have at least one variant origin of the coeliac axis or its branches and 55.4% possess the classical pattern of the hepatic arterial supply. This high percentage of variation does not lower the feasibility for the treatment. With regard to the vascular anatomy, conscientious modification under expert hands, trans-catheter ^{90}Y-SIR therapy is feasible theoretically in every individual needing it.

When compared the Hong Kong Chinese with the Europeans (reference from Michels, 1955), apparently, frequency of arterial variations is higher in the Europeans than in the Hong Kong Chinese.

From this study, there is no significant difference among the group with HCC and the group without HCC, and no gender difference in the arterial variations of the ten vessels studied.

Furthermore, when combining the three reasonings of :

1. No significant difference was observed in the group with HCC and the group without HCC.

2. No gender difference was observed.

3. A well known fact that HCC is predominantly a 'male tumour'.

One can conclude that no pattern or patterns of arterial flow predisposed to the development of HCC.

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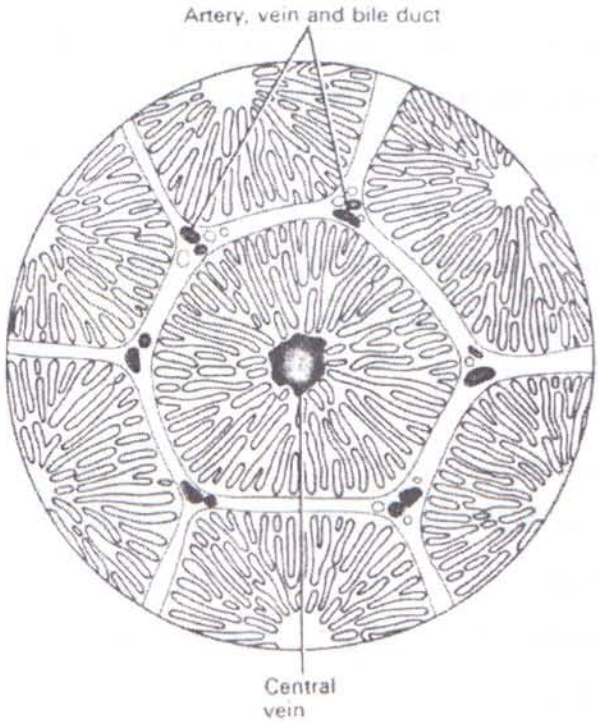
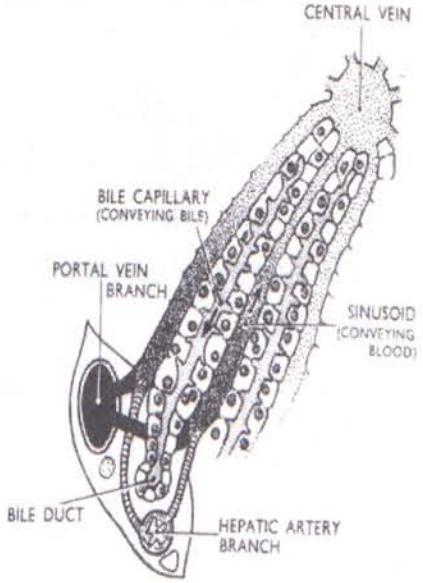
Appendix I

Schematic diagram of histological anatomy of the liver



A magnified transverse section of liver lobules
(From Anatomy and Physiology by Ross & Wilson)



Schematic drawing showing the flow of blood and bile through a liver lobule
(From Anatomy and Physiology by Ross & Wilson)

Embryology

In human fetus, organogenesis occurs between the second and the eighth week of gestation. During this period, the various organs, including the vascular system, are laid down, and differentiation begins. The primitive vascular supply is dual, with double aortas. There is a dorsal and a ventral arterial supply to the abdominal viscera. As the organs differentiate and the primitive circulation undergoes modifications, much of the double circulation is eliminated. The differing degrees of persistence of the dual circulation together with the variations in the caudal migration of the abdominal organs and rotation of the gut account for the manifestation of the many variations in the visceral circulation.

Both dorsal aortas provide ventral segmental arteries to the viscera. These give rise to the vitelline arteries (figure 2.15). At about the fourth week, fusion of the ventral roots and reduction of the segmental arteries leads to the formation of the three main ventrally oriented vessels : the coeliac axis, the superior mesenteric and inferior mesenteric arteries. By about the seventh week, the coeliac artery forms from the tenth cephalic root then migrates from the cervical region caudally to the lower thoracic-upper lumbar level. The superior mesenteric artery forms from the thirteenth cephalic root near the T1-T3 segment and the inferior mesenteric artery originates from the twenty-first or twenty-second root. Both subsequently migrates caudally to their final positions. The eleventh and twelfth root regress. The ventral anastomosis connecting the ventral segmental roots usually disappear during differentiation (figure 2.16).

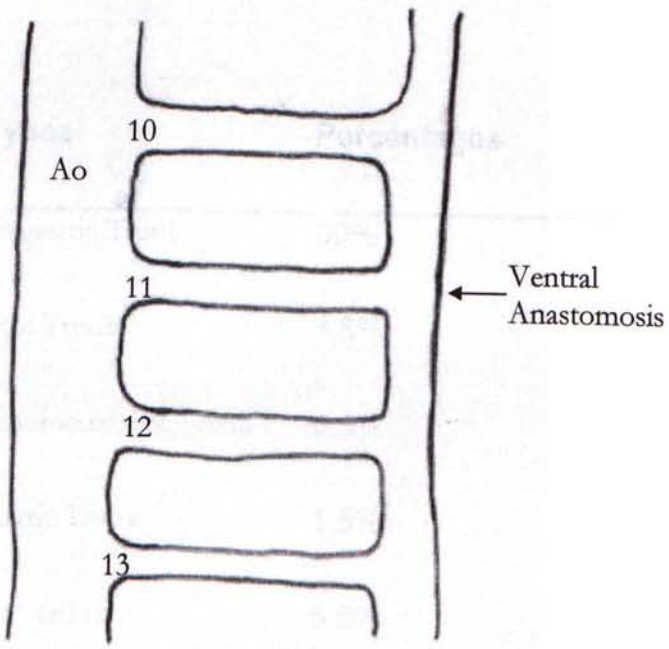


Figure IIa The primitive vascular supply - All segmental arteries and ventral anastomosis are present

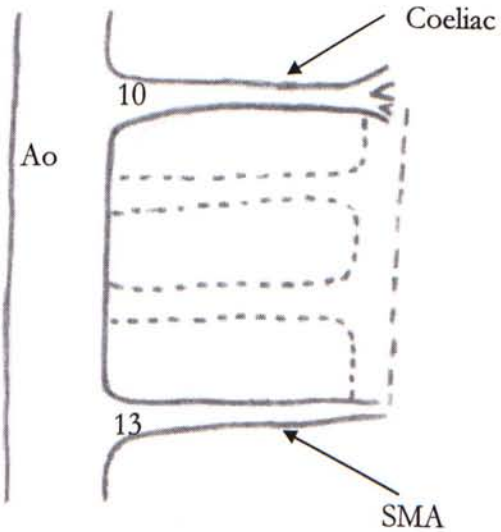


Figure IIb The 10th root forms the coeliac axis, the 13th forms the SMA, the 11th & 12th root and the ventral anastomosis regress

Percentages of occurrence of the different types of coeliac axis, by Michels' study

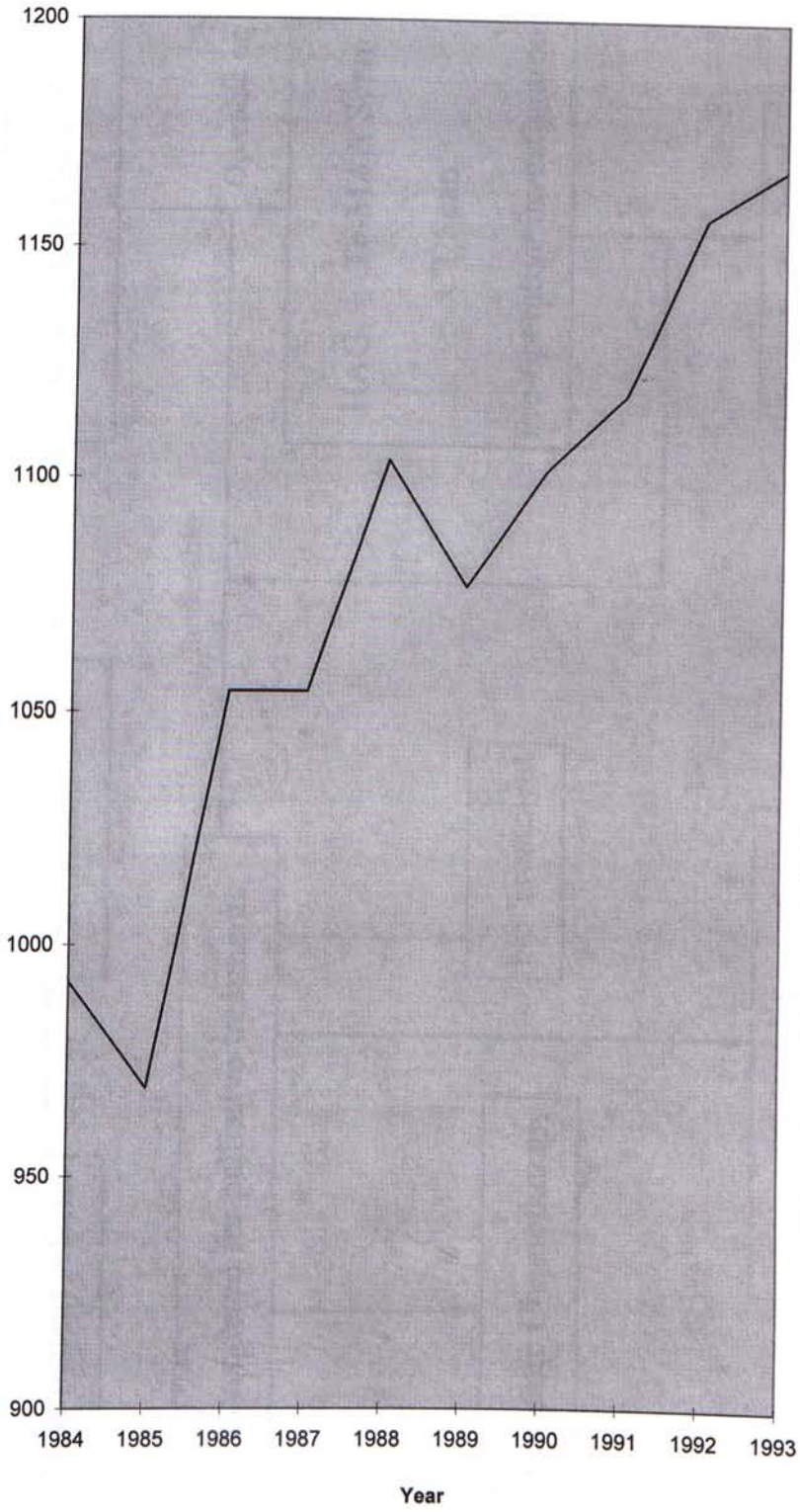
Types	Percentages
Type I. Hepatolienogastric Trunk	89%
Type II. Hepatolienal Trunk	3.5%
Type III. Hepatolienomesenteric Trunk	0.5%
Type IV. Hepatogastric Trunk	1.5%
Type V. Lienogastric Trunk	5.5%
Type VI. Coeliacomesenteric Trunk	0%
Type VII. Coeliacocolic Trunk	0%

Percentages of occurrence of the different types of the hepatic arterial blood supply, by Michels' study

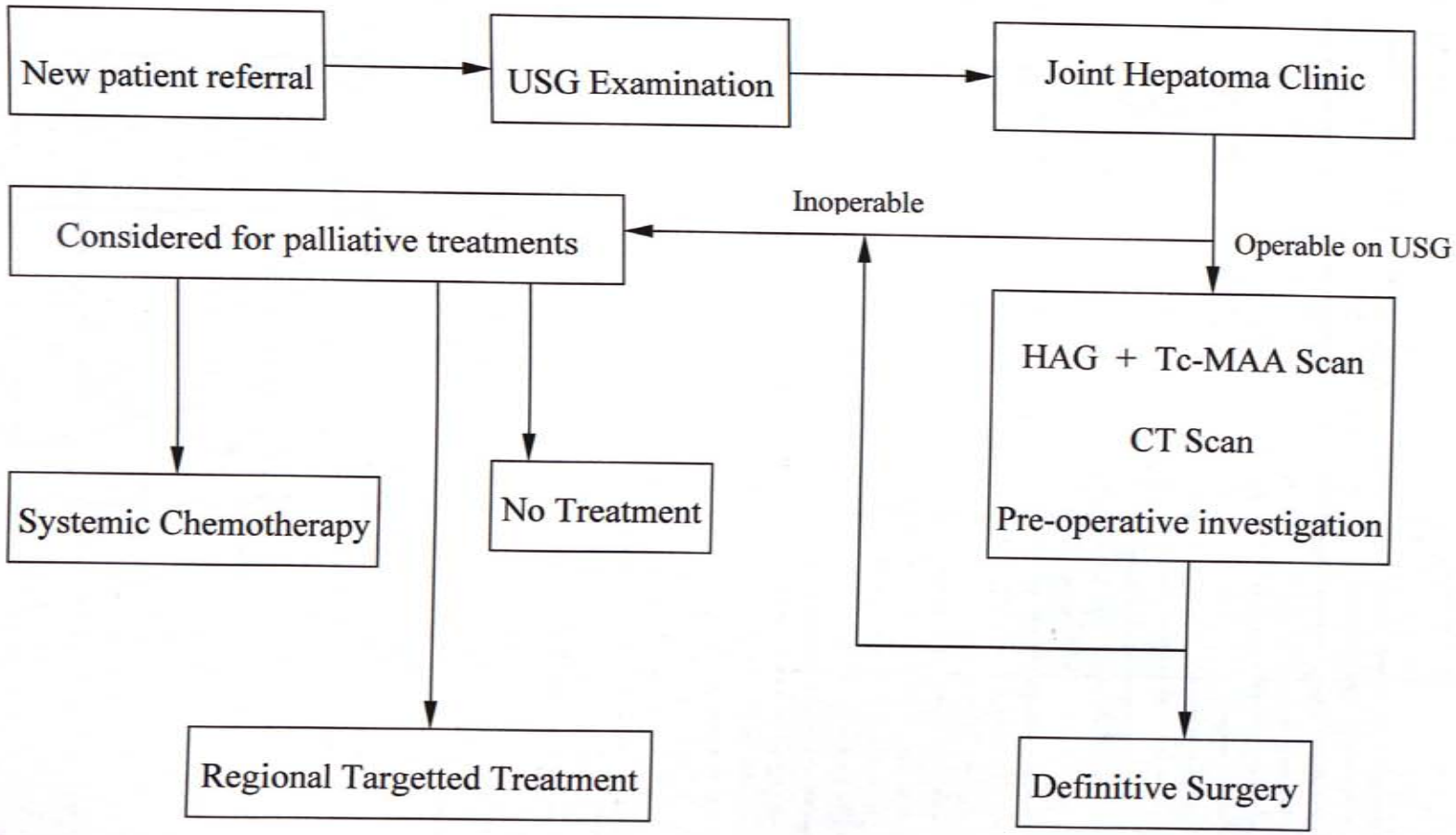
Types	Percentages
Type I.	55%
Type II	10%
Type III	11%
Type IV	1%
Type V	8%
Type VI	7%
Type VII	1%
Type VIII	2%
Type IX	4.5%
Type X	0.5%

Appendix V

No. of deaths from malignant liver cancer in H.K. (1984-1993)



Flow Chart on Management of Management of HCC in PWH, Hong Kong



Appendix VII

Comparison with Michels' study

Vessel Studied	Possible Vessel origin	% obtained from this study	% from Michels' study	p- value	
				From Chi--square test	From Fisher's Exact test
COELIAC	Ao	98.6%			
	SMA	1.1%			
	Ab	0.4%			
COMMON HEPATIC	Coeliac	97.8%			
	SMA	1.4%	2.5%		0.31000
	Ao	0.7%	1.5%		0.35000
	LG	0.0%	0.5%		0.42000
PROPER HEPATIC	CH	80.4%			
	Ab	19.6%			
RIGHT HEPATIC	PH	80.4%			
	CH	14.1%			
	SMA	4.7%	12.5%	0.00350	
	ACC fr SMA	1.1%	4.5%	0.04100	
	ACC fr GD	0.7%			
	Coeliac	0.7%	3.0%		0.06200
	ACC fr PH	0.4%			
	Ao	0.0%	2.0%		0.03100
	LG	0.0%	0.5%		0.42000
MIDDLE HEPATIC	RH	44.6%	45.0%	0.99937	
	LH	32.6%	45.0%	0.59000	
	RH & LH	14.9%			
	Ab	5.1%			
	PH	1.8%			
	CH	0.7%			
	GD	0.4%			
LEFT HEPATIC	PH	77.5%			
	CH	14.5%			
	LG	7.2%	11.5%	0.15000	
	ACC fr LG	4.0%	11.5%	0.00310	
	Coeliac	0.4%			
	Ab	0.4%			
	SMA	0.0%	2.5%		0.01300
	Ao	0.0%	1.5%		0.07000
GASTRO-DUODENAL	CH	93.1%			
	RH	5.1%	7.0%	0.49000	
	LH	1.4%	11.0%	0.00002	
	Coeliac	0.4%	1.5%		0.20000
	SMA	0.0%	1.0%		0.18000
	MH	0.0%	1.0%		0.18000
RIGHT GASTRIC	PH	42.0%			
	LH	35.5%	40.5%	0.30000	
	RH	10.1%	5.5%	0.09800	
	CH	4.3%			
	GD	3.6%	8.0%	0.06200	
	Ab	2.2%			
	MH	1.1%	5.0%	0.02100	
	BIF of PH	0.7%			
	BIF of CH	0.4%			
LEFT GASTRIC	Coeliac	94.6%	90.0%	0.08810	
	Ao	2.2%	2.5%		0.52000
	BIF of Coeliac	1.8%			
	Ab	1.4%			
SPLENIC	Coeliac	99.3%			
	Ao	0.4%			
	SMA	0.4%			

Data not available

Appendix VIII

Comparison of the group with HCC with the group without HCC

Vessel Studied	Possible Vessel origin	No. with HCC	No. without HCC	p- value	
				From Chi--square test	From Fisher's Exact test
COELIAC	Ao	163	109		0.48
	SMA	3	0		0.22
	Ab	0	1		0.40
COMMON HEPATIC	Coeliac	162	108		0.55
	SMA	3	1		0.48
	Ao	1	1		0.64
PROPER HEPATIC	CH	132	90	0.75	
	Ab	34	20	0.75	
RIGHT HEPATIC	PH	131	91	0.53	
	CH	25	14	0.71	
	SMA	9	4	0.69	
	ACC fr SMA	2	1		0.65
	ACC fr GD	1	1		0.64
	Coeliac	1	1		0.64
	ACC fr PH	1	0		0.60
MIDDLE HEPATIC	RH	73	50	0.91	
	LH	60	30	0.16	
	RH & LH	19	22	0.07	
	Ab	9	5	0.96	
	PH	3	2		0.66
	CH	1	1		0.64
	GD	1	0		0.60
LEFT HEPATIC	PH	127	87	0.72	
	CH	27	13	0.39	
	LG	11	9	0.93	
	ACC fr LG	7	4		0.54
	Coeliac	1	0		0.60
	Ab	0	1		0.40
GASTRO-DUODENAL	CH	152	105		0.16
	RH	10	4		0.28
	LH	3	1		0.48
	Coeliac	1	0		0.60
RIGHT GASTRIC	PH	68	48	0.75	
	LH	59	39	0.91	
	RH	20	8	0.28	
	CH	6	6		0.33
	GD	5	5		0.36
	Ab	4	2		0.55
	MH	2	1		0.65
	BIF of PH	1	1		0.64
	BIF of CH	1	0		0.60
LEFT GASTRIC	Coeliac	159	102	0.41	
	Ao	3	3		0.45
	BIF of Coeliac	2	3		0.31
	Ab	2	2		0.52
SPLENIC	Coeliac	166	108		0.16
	Ao	0	1		0.40
	SMA	0	1		0.40

Not applicable

Appendix IX

Comparison of the male and female group

Vessel Studied	Possible Vessel origin	No. of Male subject	No. of Female subject	p- value	
				From Chi--square test	From Fisher's Exact test
COELIAC	Ao	193	79		0.26
	SMA	3	0		0.36
	Ab	1	0		0.71
COMMON HEPATIC	Coeliac	192	78		0.45
	SMA	3	1		0.68
	Ao	2	0		0.51
PROPER HEPATIC	CH	158	64	0.99	
	Ab	39	15	0.99	
RIGHT HEPATIC	PH	157	65	0.75	
	CH	29	10	0.8	
	SMA	9	4		0.54
	ACC fr SMA	1	2		0.2
	ACC fr GD	1	1		0.49
	Coeliac	2	0		0.51
	ACC fr PH	1	0		0.71
MIDDLE HEPATIC	RH	89	33	0.7	
	LH	68	22	0.35	
	RH & LH	24	17	0.07	
	Ab	9	5		0.37
	PH	4	1		0.56
	CH	1	1		0.49
	GD	1	0		0.51
LEFT HEPATIC	PH	153	62	0.99	
	CH	32	8	0.26	
	LG	12	8	0.36	
	ACC fr LG	5	6		0.06
	Coeliac	1	0		0.71
	Ab	0	1		0.29
GASTRO-DUODENAL	CH	180	77	0.12	
	RH	12	2		0.18
	LH	4	0		0.26
	Coeliac	1	0		0.71
RIGHT GASTRIC	PH	86	30	0.47	
	LH	67	31	0.5	
	RH	22	6	0.5	
	CH	8	4		0.47
	GD	8	2		0.42
	Ab	3	3		0.23
	MH	2	1		0.64
	BIF of PH	0	2		0.08
	BIF of CH	1	0		0.71
LEFT GASTRIC	Coeliac	186	75		0.56
	Ao	3	3		0.23
	BIF of Coeliac	4	1		0.56
	Ab	4	0		0.26
SPLENIC	Coeliac	196	78		0.49
	Ao	0	1		0.29
	SMA	1	0		0.71

Not applicable

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